THE Journal Kansas Medical Society

MAY
1957

VOL LVIII
NO V

MAY 13 1957

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CLINICAL EXPERIENCE INDICATES FEWER RESISTANT STAPHYLOCOCCI

CHLOROMY

As clinical reports on resistance of common pathogens to antimicrobial therapy gain increasing prominence,[1-5] need for broad-spectrum antibiotic therapy to which resistance is less likely to develop becomes even more apparent. Particularly troublesome are the staphylococci, which often fail to respond not only to commonly used antibiotic therapy but also to agents more recently introduced.[6-10]

CHLOROMYCETIN (chloramphenicol, Parke-Davis) has maintained most of its original effectiveness against strains of staphylococci and against other sensitive pathogens.[2-4,11-15] "The fact that so few strains were found to be resistant to chloramphenicol [CHLOROMYCETIN] made it possible for the clinicians to turn to this antibiotic when such a large proportion of strains was observed to be highly resistant to the other commonly used antibiotics."[2]

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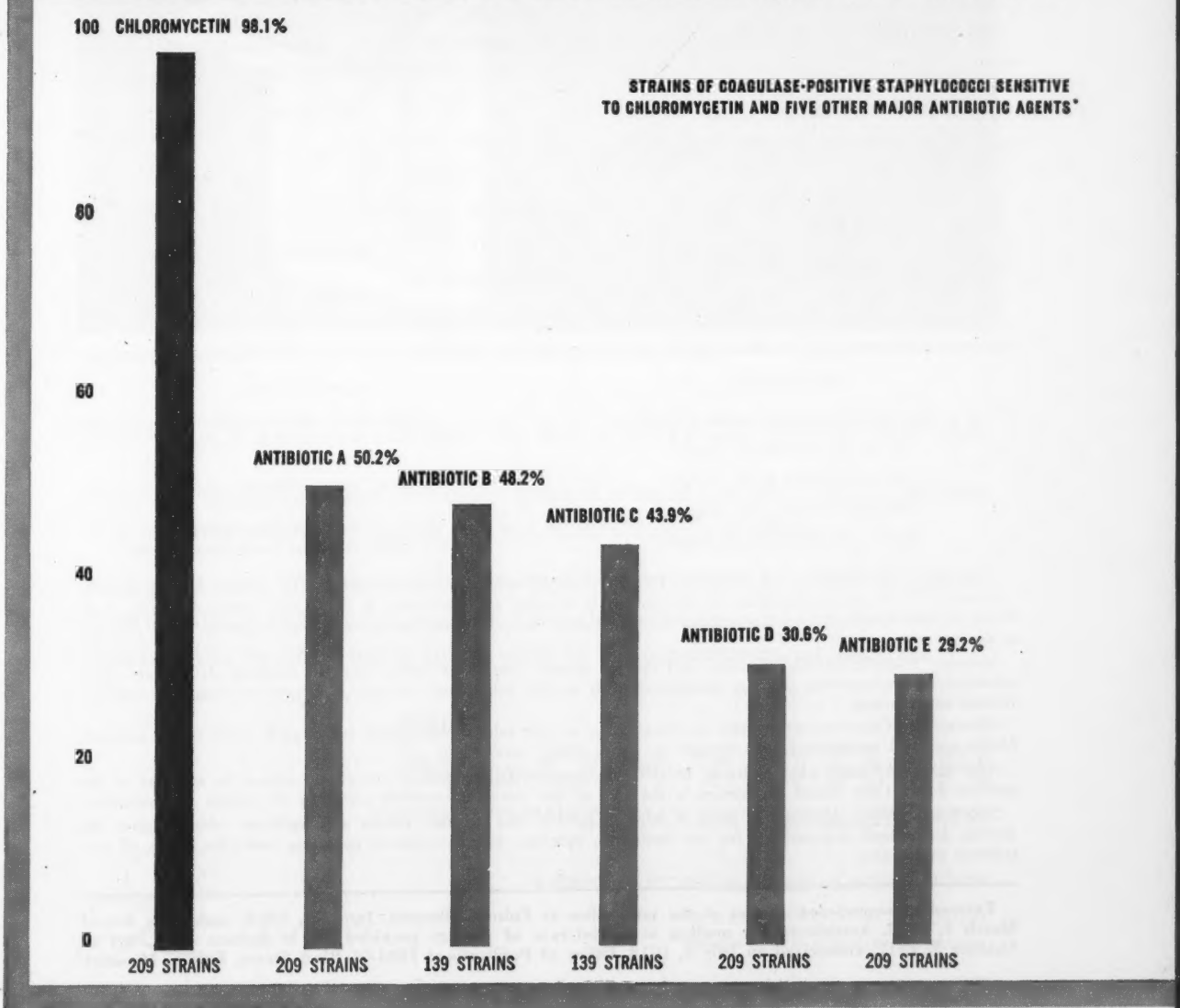

*This graph is adapted from Spink.[2]

THE JOURNAL
of the
KANSAS MEDICAL SOCIETY

Volume LVIII MAY, 1957 Number 5

TABLE OF CONTENTS

Original Articles

Myotonia—Report of a Case in Transition between Myotonia Congenita and Myotonia Dystrophica—Harry H. White and Charles M. Poser, M.D., Kansas City 293

Medical Services—Distribution of Physicians and General Hospital Beds in Kansas in 1955—E. V. Thiehoff, M.D., Kansas City . 297

Cryptococcosis—Treatment of Two Cases With Stilbamidines—Wirt A. Warren, M.D., G. Gayle Stephens, M.D., and Ernest W. Crow, M.D., Wichita 301

Abdominal Pregnancy—Report of Successful Management of One Case—L. W. Patzkowsky, M.D., Kiowa, M. D. Christensen, M.D., Kiowa, and J. F. Simon, M.D., Alva, Oklahoma 304

Clinicopathological Conference 316

Pulmonary Edema—A Study of Its Pathogenesis and Review of the Literature—Darrell D. Fanestil, Kansas City 328

Editorials

Uniform Narcotics Act 307

Soviet Medicine 308

Heedless Horsepower 308

Miscellaneous

President's Page 306

Just Browsing 312

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Ownership: The Journal is a non-profit publication owned and published monthly by the Kansas Medical Society.

Subscription: A year's subscription to the Journal is included in membership in the Kansas Medical Society, with $2.00 of each member's dues apportioned to the Journal. Rates to others, except in foreign countries, $4.00 per year or 60c per copy.

Material: Scientific articles, editorials, and data of general interest are invited from all members. Articles are to be submitted on condition that they are contributed solely to this publication. A right is reserved to reject any material deemed unsatisfactory.

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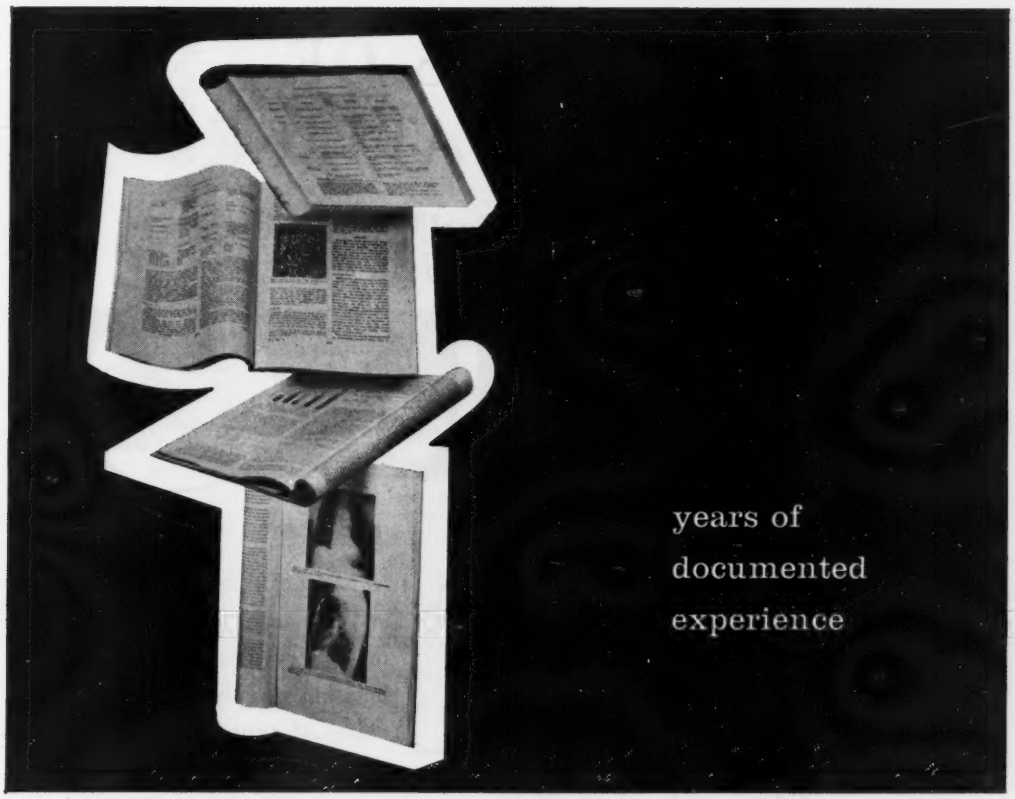

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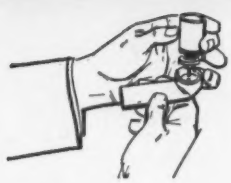

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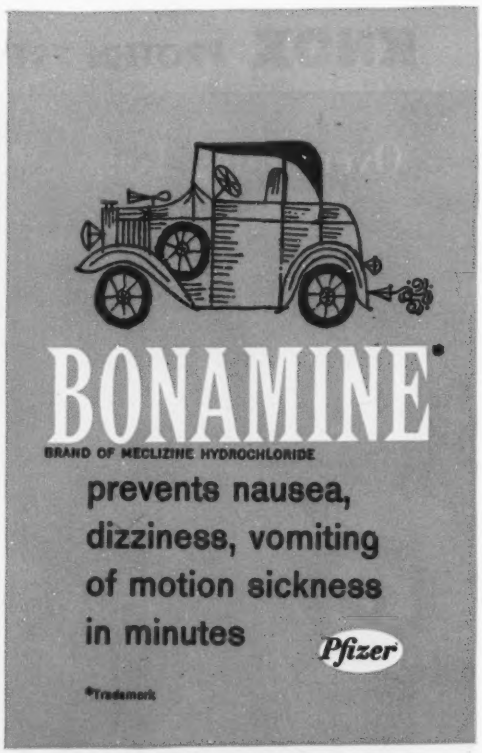

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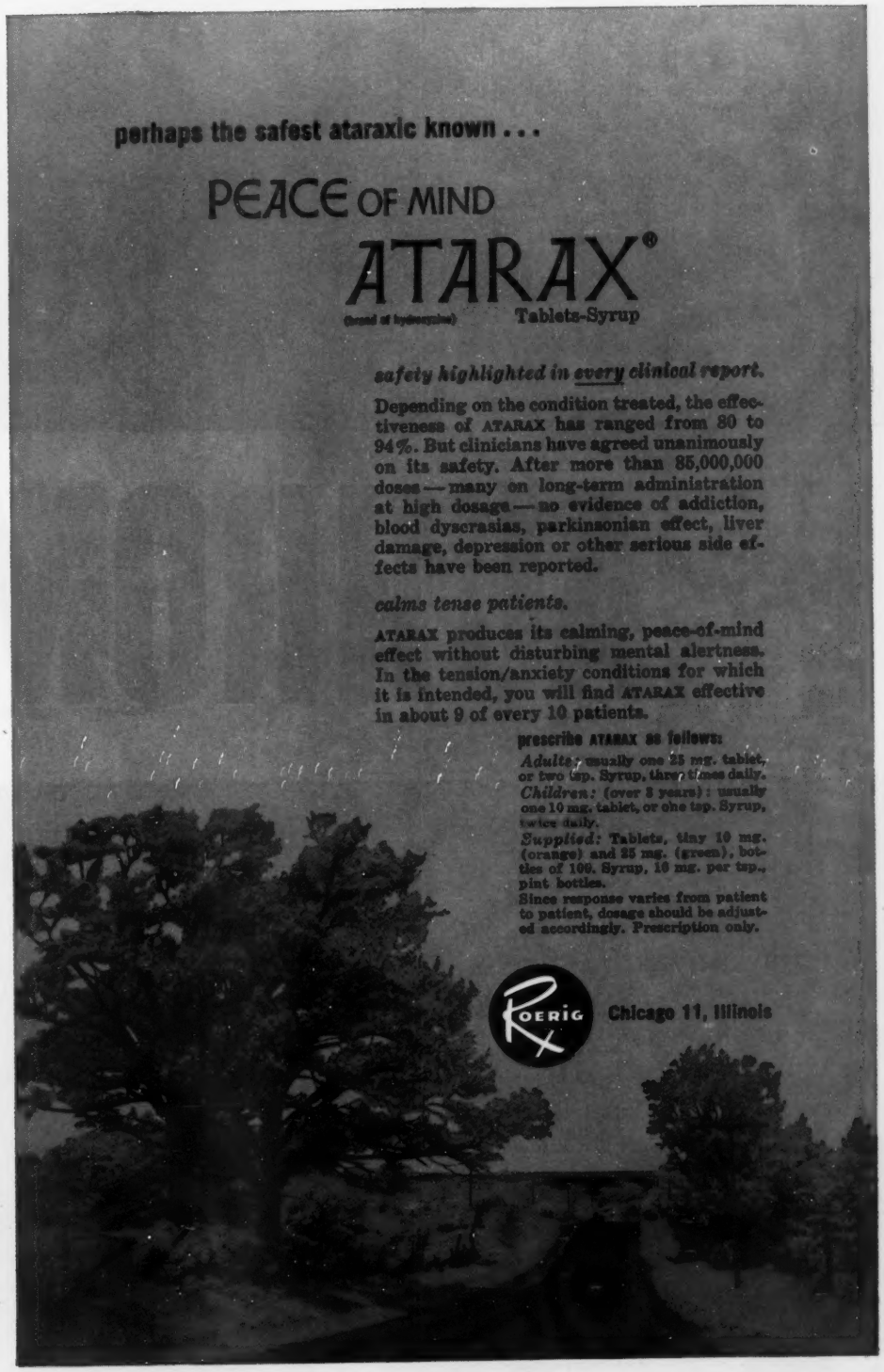
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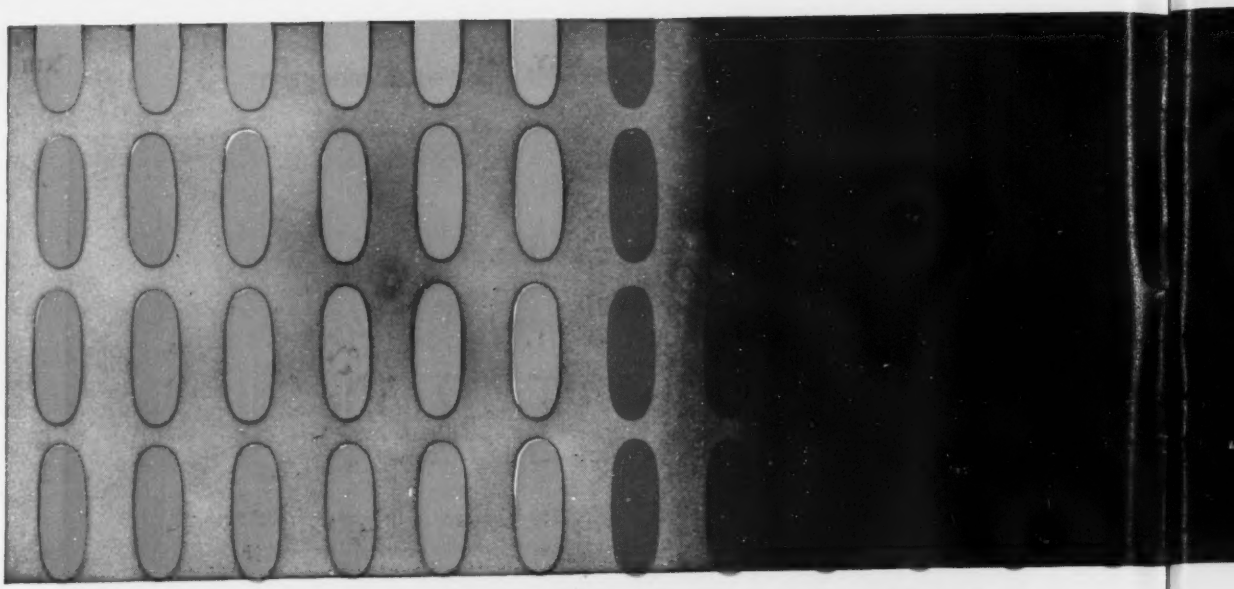

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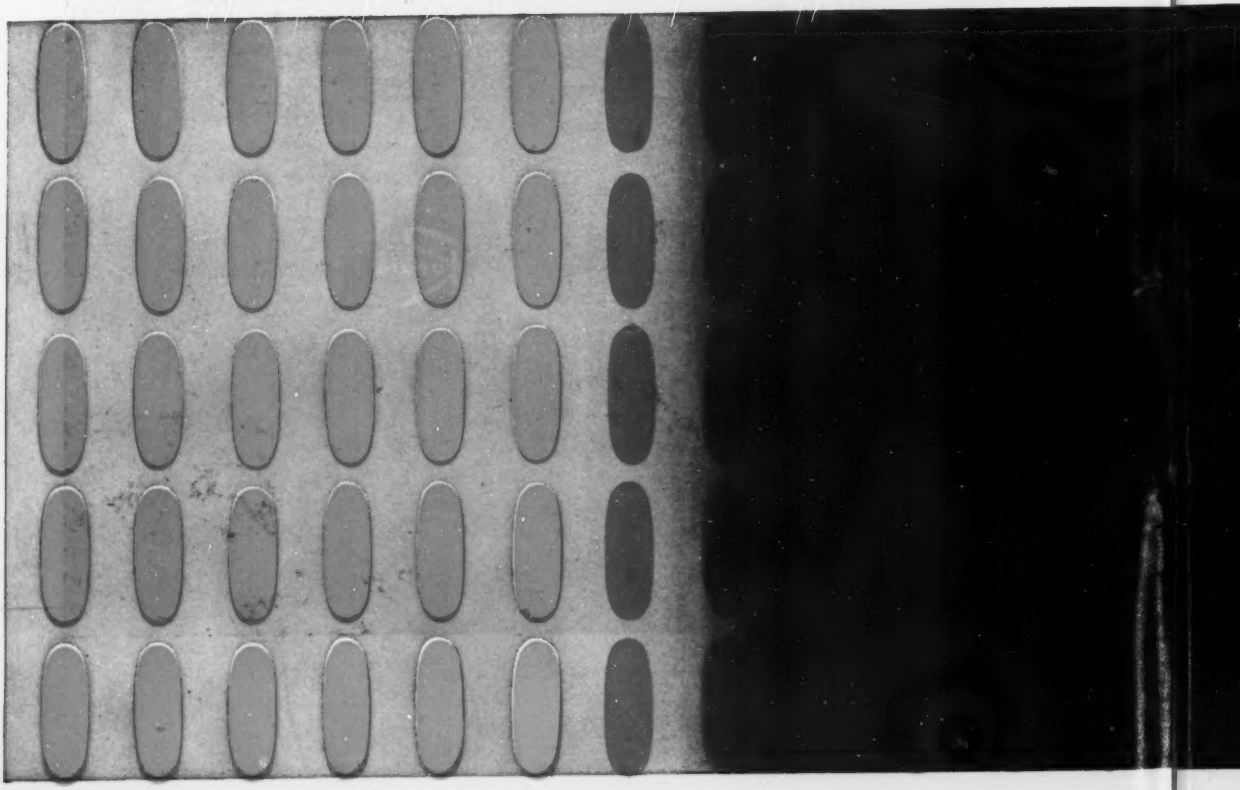

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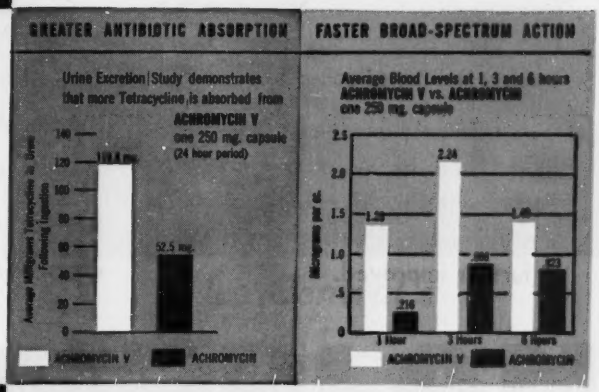

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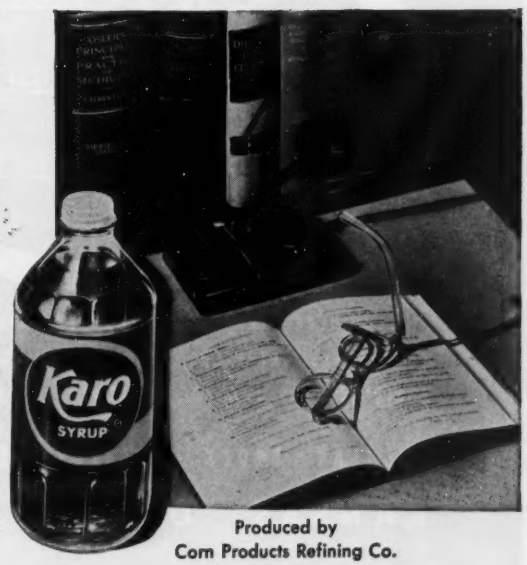

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Thimann, J. and Gauthier, J.W.: Quart. J. Stud. Alcohol. **17**: *19, 1956.*

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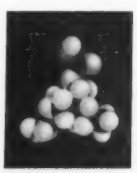

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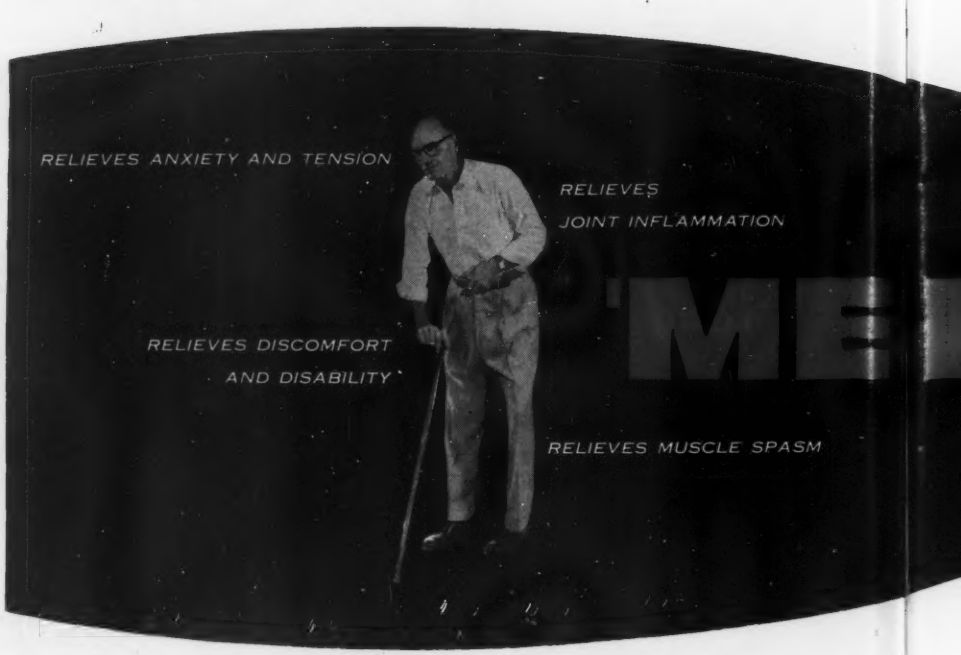

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2. *Meprobamate*—the newest and safest of the muscle-relaxant tranquilizers for profound relaxation of skeletal muscle in spasm.

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INDICATIONS: A wide variety of conditions, in which four symptoms predominate: *a*) inflammation *b*) muscle spasm *c*) anxiety and tension *d*) discomfort and disability; i.e., rheumatoid arthritis, rheumatoid spondylitis (Marie-Strümpell disease), Still's disease, psoriatic arthritis, osteoarthritis, bursitis, synovitis, tenosynovitis, myositis, fibrositis, fibromyositis, neuritis, acute and chronic low back pain, acute and chronic primary and secondary fibrositis and torticollis, intractable asthma, respiratory allergies, allergic and inflammatory eye and skin disorders (as maintenance therapy in disseminated lupus erythematosus, periarteritis nodosa, dermatomyositis and scleroderma).

Therapeutic benefits of MEPROLONE compared with traditional antiarthritics.

	relieves pain	suppresses inflammation	relaxes muscle	eases anxiety	imparts sense of well-being
Salicylates	✓	✓			
Muscle relaxants			✓[1]		
Tranquilizers				✓[1]	
Steroids	✓	✓			✓
MEPROLONE	✓	✓	✓	✓	✓

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Volume LVIII **MAY, 1957** **No. 5**

Myotonia

Report of a Case in Transition between Myotonia Congenita and Myotonia Dystrophica

HARRY H. WHITE *and* **CHARLES M. POSER, M.D.,** *Kansas City*

Since 1886, when Erb[1] wrote his classic description of the myotonic reaction, a number of disease entities have been delineated which have as their distinguishing feature a myotonic reaction of various muscle groups. Three main diseases of this type have been recognized: myotonia dystrophica of Steinert,[1a] myotonia congenita of Thomsen[1b] and paramyotonia of Eulenberg.[1c]

Myotonia dystrophica (Steinert's disease) is also characterized by what seem to be endocrinopathic features such as frontal baldness, testicular atrophy, and cataracts, while in myotonia congenita (Thomsen's disease) there is hypertrophy of muscles. Although it is well known that any type of myotonia will be aggravated by cold, Eulenberg's paramyotonia has as its only characteristic a myotonic reaction produced by cold.

Certain neurologists, however, have been reluctant to separate the three entities, and evidence has been brought forward to support the contention that they are merely variant manifestations of a single disease process. The transitional cases reported in the literature reinforce this view.

The purpose of this paper is to report a case that is felt to represent a transitional stage between myotonia dystrophica of Steinert and myotonia congenita of Thomsen.

Case Report

W. M., a 35-year-old salesman, first entered a naval hospital in California in February, 1944, with the complaint of "stiffness" of the body and progressive enlargement of the calf muscles for a month. While riding on a train, he had attempted to rise from his seat. He found he was unable to straighten out, and his muscles had become "stiff." He said he had had transitory episodes of stiffness for the past two years, and that he was unable to run unless he began by walking slowly and then gradually increasing his speed. He had noticed that he had difficulty in taking hold of things until after his fingers were "worked a while," at which time "full power comes to them." He felt his condition was more severe during cool weather.

In the case reported myotonia congenita is suggested by the presence of hypertrophy of the calf muscles. The diagnosis of myotonia dystrophica is indicated by small sternomastoids, cataracts, distribution of the myotonia, generalized weakness, and decreased urinary 17-ketosteroids. Long-term abolition of the myotonic reaction through the use of cortisone and prednisone is reported.

Physical examination revealed enlargement of the calf muscles of both legs. No mention was made of myotonic contractions. The rest of the physical examination was negative. The diagnosis of myotonia (etiology undetermined) was made. The patient received 5 grains of quinine three times a day for a period of two days with no subjective improvement,

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and he then was dismissed to return to active military duty.

The patient re-entered the same hospital in March, 1944, with the same complaints. His symptoms had progressed to a degree that he was handicapped in performance of his duties. A biopsy of his gastrocnemius muscle showed muscle fibers which were thicker than normal, many areas in which the longitudinal fibrils could not be distinguished, an increase in the number of sarcolemmal nuclei, preservation of cross striations, no fatty changes, and one area disclosing a small collection of lymphocytes. The pathological diagnosis of the biopsy specimen was "changes compatible with myotonia congenita."

After his case was reviewed he was given a medical discharge in May, 1944. Shortly thereafter he noted occasional difficulty in opening his clenched fist.

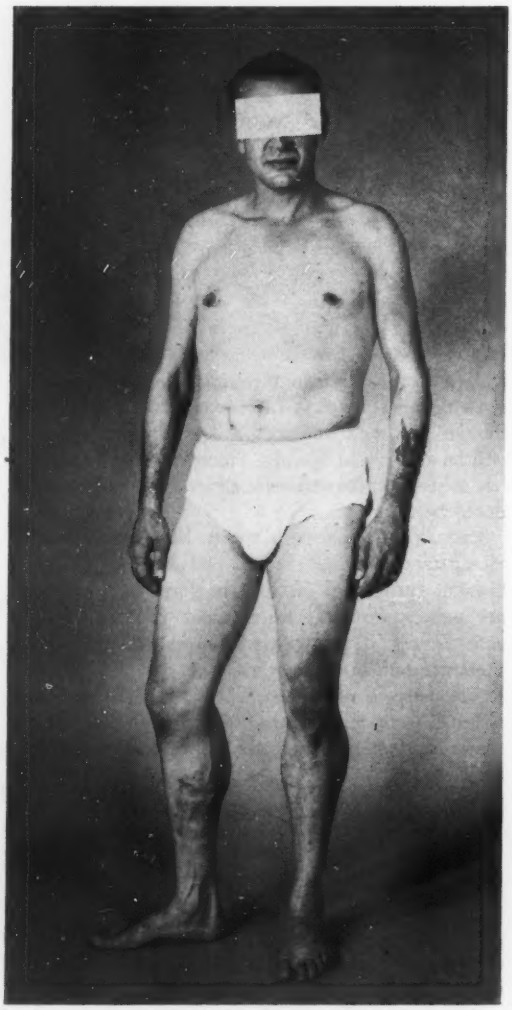
From 1944 to 1951 there were no subjective changes in his condition. From 1951 to 1956 he noticed increasing weakness in carrying out his normal business activities, and in June of 1956 he entered the hospital with a complaint of "stiffness" of his muscles.

The family history was as follows: The patient's father died at 55 of cancer of the liver, and his mother died at 58 of heart trouble. The patient's father had one brother who died of a stroke and two sisters, one of whom died of unknown causes; the other is living and well. His mother had two sisters and one brother, all living and well. The patient had had four siblings, all dead. One brother died at 6½ of blood poisoning; one sister died at 28 in childbirth; one sister died at 17 of a head injury, and another sister died at 24 of an internal hemorrhage. There was no family history of early baldness, cataracts, sterility, or other endocrine dysfunction. The patient had two children, ages 6 and 11, by his first wife, both living and well. He married a second time in 1954 and had thus far been unsuccessful in attempts to have children with this wife.

On neurologic examination the patient was seen to be a well-developed, well-nourished white male with no frontal baldness and facies not strikingly of the myopathic type. He had obvious difficulty in starting to walk after being in the sitting position, but when he began to walk his gait was not remarkable. When he stood on his toes for a short time he had some difficulty in bringing his heels down to the floor.

No abnormalities were noted on fundoscopic examination, and there were no obvious lenticular opacities. Slit-lamp examination, however, revealed punctate lenticular opacities in the peripheral portions of the cortex of each lens. The media were grossly clear. The pupils were round, regular, and equal, and they responded to light and accommodation. Extraocular movements were normal and there was no nystagmus. There was no evidence of facial weakness, but in contrast to the man's rather mesomorphic appearance there were slight bilateral weakness and some atrophy of the sternomastoid muscles. The patient had generalized muscular weakness, most pronounced distally, in all extremities. No atrophy or fasciculations were present. There was striking enlargement of both calf muscles, which measured 38½ cm. in circumference in their largest diameter. Spontaneous myotonic reactions were observed in the hands and percussion myotonia was elicited in the tongue and thenar eminences.

Superficial and deep-tendon reflexes were active and equal bilaterally. There were no pathologic reflexes. Sensory examination and testing of cerebellar functions were entirely normal. The heart was not enlarged. There were no thrills or murmurs. The patient had a regular sinus rhythm with a pulse



of 80 and a blood pressure of 110/70. There was no testicular atrophy, and the body hair distribution was of the male type. The thyroid was not enlarged to palpation.

Hemogram, urinalysis, and erythrocyte sedimentation rates were within normal limits. Roentgenograms of the skull and the chest were normal. Results of other laboratory examinations are summarized in Table I.

The patient was started on procaine amide, 250 mgm. three times a day, and cortisone, 50 mgm. four times a day. On this regimen there was distinct subjective and objective improvement of his myotonia. The myotonic contractions in the hands almost completely disappeared, and the patient was able to rise from the sitting position more quickly and fatigued less rapidly when walking. Procaine amide was discontinued after two weeks, and the patient continued to improve on cortisone alone.

After three weeks, cortisone was discontinued and the patient was given prednisone, 5 mgm. four times a day. He was discharged to his home on this medication, and when he was seen again, ten weeks later, the myotonic reaction was still abolished.

TABLE I

LABORATORY EXAMINATIONS

1. *Blood*	
Fasting blood sugar:	100 mg. per 100 ml.
Non-protein nitrogen	34.4 mg. per 100 ml.
Serum albumin	4.4 g. per 100 ml.
Serum globulin	2.3 g. per 100 ml.
Serum creatinine	1.2 mg. per 100 ml.
Total cholesterol	270.0 mg. per 100 ml.
Cholesterol esters	138.0 mg. per 100 ml.
Serum sodium	137 mEq. per 100 ml.
Serum potassium	5.0 mEq. per 100 ml.
Serum chloride	96 mEq. per 100 ml.
Serum carbon dioxide	24.2 mEq. per 100 ml.
Serum calcium	4.4 mEq. per 100 ml.
Serum phosphorus	1.8 mEq. per 100 ml.
Thymol turbidity	2 units
Protein bound iodine	10.0 microgr. "
Bromsulphonpthalein retention	0%
Alkaline phosphatase	2.9 Bodansky units
Serum electrophoresis:	slight increase in alpha-2 globulins
2. *Urine*	
Creatine excretion	0
Creatinine excretion	1.7 gms. per 24 hrs.
17-Ketosteroids excretion	12.8 mg. per 24 hrs.
3. *Cerebrospinal fluid*	
Cells	0
Total protein	35 mg. per 100 ml.
Colloidal gold curve	0011110000
4. *Miscellaneous*	
Radioactive iodine uptake	26%
Sperm count: good motility—	120-140 million
Electrocardiogram:	normal
Biopsy, gastrocnemius muscle:	normal muscle

Discussion

Several features of the mode of onset in this case are unusual. First, there were several short episodes of transient "stiffness" which were minimal in duration and intensity; second, there occurred a sudden and severe episode of myotonia for which the patient first sought medical attention; last is the early complaint of enlarged calf muscles. The usual case of myotonia manifests itself by a slow, progressive, and persistent myotonia with atrophy or hypertrophy becoming noted somewhat later during the course of the disease.

In addition to the "classical" features of myotonia dystrophica such as cataracts, frontal baldness, muscular atrophy, sterility, and the myopathic facies, certain electrocardiographic abnormalities have been reported as well as low basal metabolic rates and a decreased excretion of urinary 17-ketosteroids, evidence to support the belief that this disease is multisystemic in its involvement, and strongly suggestive of an endocrinopathy, the nature of which is still obscure.

In the present case, early cataracts, small sternomastoid muscles, the decreased urinary 17-ketosteroids, generalized muscular weakness, and the distribution of the myotonia suggest the diagnosis of myotonia dystrophica. Against such a diagnosis are the absence of frontal baldness, the absence of the characteristic facies, the lack of testicular atrophy, a normal electrocardiogram, no indication of endocrinopathy, and a negative family history. Adams, Denny-Brown, and Pearson[7] stated that increased creatine and decreased creatinine excretion in the urine are the most constant biochemical changes in cases of myotonia dystrophica. The normal creatine-creatinine excretion ratio in this case seems fairly good evidence against any significant amount of muscle atrophy.

One of the outstanding signs in this case is the striking hypertrophy of the calf muscles, the characteristic feature of Thomsen's myotonia congenita. Myotonia congenita, however, usually manifests itself in early childhood, and frequently more than a single member of one family is affected. However, Adams, Denny-Brown, and Pearson[7] stated that only one-fourth of the reported cases are hereditary, and in them the disorder appears as Mendelian dominant. Our patient was affected in early manhood, and there was no similar affliction of his siblings.

In some instances myotonia dystrophica may undoubtedly progress for a while without showing some of the characteristic features of the disease. However, in our patient the disease has been present for at least 12 years, and only minimal dystrophic or endocrinopathic manifestations could be demonstrated. On the other hand, early hypertrophy of the calves is strongly suggestive of the type of muscular involvement seen in myotonia congenita. Our case may deserve classification simply as an abortive case of myotonia dystrophica, but, in the presence of a well-developed myotonic reaction and obvious muscular hypertrophy, it would seem not illogical to consider it a transitional case between two closely related disease entities.

Cases similar to the present one have been reported before. For example, A. M. (family 121 of Maas and Paterson[8]) was a man of pyknic build, had generalized myotonia and hypertrophy of the deltoids, pectorales, and thigh muscles. Such features are characteristic of myotonia congenita, but the patient also had myopathic facies and pronounced wasting and weakness of other muscles, features which are characteristic of myotonia dystrophica.

Another case in this series was that of J. G. Goo (family 56), who had suffered from stiffness since infancy and possessed the typical "Herculean body build" frequently described in myotonia congenita. He subsequently developed wasting of the sternomastoids and facial muscles, at which time his illness was rediagnosed as myotonia dystrophica.

Arthur R. Bu (family 19) was believed to have myotonia congenita when he was 22, but four years later he showed wasting of certain facial muscles and the left sternomastoid, muscular weakness, frontal baldness, myopathic facies, and lenticular opacities, at which time there was no question as to the diagnosis of myotonia dystrophica.

It was well established that quinine had no effect on our patient's disease. His lack of response to procaine amide may be due to the fact that he was given a maximum of only 750 mg. per day, while the favorable results reported by Geschwind and Simpson[9] in nine myotonic patients were obtained by administering daily amounts of between 2,000 and 5,000 mg.

The merits of steroid therapy in myotonia are still not completely evaluated. In 1950 Milhorat[10] first gave ACTH to two patients and reported subjective improvement. In the same year Shy, Brendler, Rabinowitch, and McEachern[11] were the first to report an abolition of the myotonic reaction with cortisone in two patients. In 1954 Garai[12] reported a good response to ACTH in two of four patients. In one of the patients this response lasted six to eight weeks after the last administration of ACTH. Holland and Hill[6] obtained a transitory abolition of the myotonia by giving intravenous ACTH to one patient.

On the other hand, Trevathan and Hussar,[3] in 1953, reported no improvement in one patient who was given at first as much as 200 mg. of cortisone daily, followed by 100 mg. daily of ACTH, nor in another patient who received 100 mg. of cortisone daily.

University of Kansas Medical Center
39th and Rainbow
Kansas City 12, Kansas

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Voluntary health insurance now pays more than 25 per cent of the total annual expenses for all personal health services incurred by both insured and uninsured persons in this country, Health Information Foundation reports.

Medical Services

Distribution of Physicians and General Hospital Beds in Kansas in 1955

E. V. THIEHOFF, M.D., ***Kansas City***

Specialization of Kansas Physicians

A study of the distribution of physicians and their services in Kansas would not be complete unless attention is given to the relative distribution of the services of specialists and general practitioners. In dealing with this matter, physicians have been divided into three categories:

1. Certified specialists
2. Non-certified specialists
3. General practitioners

The designation of specialist is entirely the decision of the individual physician. So we find a wide range of criteria as to what constitutes a specialist. This is true in the non-certified group of specialists. With regard to certified specialists there are definite standards as to training and limitation of practice, established by specialty boards.

The list of physicians now in Kansas who are certified specialists was first determined by reference to the *Directory of Medical Specialists,* Volume 6, 1953 and then, more recently, to Volume 7, 1955, of that directory. The list of non-certified specialists was obtained from records of the Kansas Medical Society and from information supplied by physicians on their re-registration cards submitted to the Kansas State Board of Medical Registration and Examination. All other physicians, not listed as certified or non-certified specialists, have been classed as general practitioners, although undoubtedly there are many of these latter physicians who would consider themselves as non-certified specialists.

There is no general standard established as to the desirable proportion of specialists to general practitioners. This would necessarily vary from place to place, depending upon various factors. So we see there are definite limitations in the study of statistics related to specialization of physicians.

Table XV indicates that in Kansas we have gone a long way toward specialization; 40.5 per cent of the physicians in the state today regard themselves as specialists, 16.15 per cent by right of certification and 24.36 per cent by self designation. There are more in the latter group than in the former. And 59.5 per cent of physicians in the state are classed as general practitioners. It is not known whether or not this is a desirable proportion.

It has been the consensus that the services of physicians, particularly specialists, are more readily available in the larger communities. Let us see what the situation is in Kansas.

TABLE XV

DISTRIBUTION OF PHYSICIANS BY SPECIALIZATION

Status as to Specialty	*Number*	*Per Cent*
Certified	378	16.15
Non-Certified	570	24.36
General Practice	1392	59.49
TOTAL	2340	100.00

This is Part II of a three-part series. Part I appeared in the April issue of the Journal, and Part III will follow in June.

TABLE XVI

DISTRIBUTION OF PHYSICIANS BY SIZE OF COMMUNITY

Size of Community	*Number of Physicians* SPECIALTY CERTIFIED	NON-CERTIFIED	GENERAL PRACTICE	TOTAL
Large	294	412	450	1156
Intermediate	75	129	523	727
Small	9	29	419	457
TOTAL	378	570	1392	2340

As was to be expected, there are many more physicians practicing in large communities (30,000 or more) than in small communities (less than 5,000). However, upon breaking these figures down as to

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specialist and general practitioners, we see that specialists are predominantly in the large cities. General practitioners are about evenly divided between large and small communities, but there are more in cities of intermediate size (5,000 to 30,000 population). Let us see what these figures mean percentage-wise.

The large cities of Kansas (over 30,000 population) have 24.6 per cent of the state's population, but have 49.4 per cent of all physicians, 77.78 per cent of all certified specialists, 72.28 per cent of all non-certified specialists, and 32.33 per cent of the general practitioners. Of all physicians practicing in the large cities, 25 per cent are certified, 36 per cent are non-certified, and 39 per cent are in general practice, making a total of 61 per cent who consider themselves as specialists compared with 39 per cent who are general practitioners. So there appears to be a disproportionate number of specialists.

TABLE XVII

PERCENTAGE DISTRIBUTION OF PHYSICIANS BY SPECIALTY AND SIZE OF COMMUNITY

Size of Community	*Status as to Specialty* CERTIFIED	NON-CERTIFIED	GENERAL PRACTICE	TOTAL
Large	77.78	72.28	32.33	49.40
Intermediate	19.84	22.63	37.57	31.07
Small	2.38	5.09	30.10	19.53
TOTAL ...	100.00	100.00	100.00	100.00

TABLE XVIII

DISTRIBUTION OF PHYSICIANS BY PRINCIPAL CITIES

Place	*Physicians* SPECIALTY CERTIFIED	NON-CERTIFIED	GENERAL PRACTICE	TOTAL
	NUMBERS			
Kansas City..	82	147	128	357
Topeka	86	121	105	312
Wichita	102	137	152	391
Rest of State.	108	165	1007	1280
TOTAL	378	570	1392	2340
	PER CENT			
Kansas City..	21.68	25.79	9.2	15.26
Topeka	22.74	21.23	7.54	13.33
Wichita	26.97	24.03	10.92	16.71
Rest of State.	28.61	28.95	72.34	54.70
TOTAL 100.00	100.00	100.00	100.00	

The cities of intermediate size (5,000 to 30,000 population) have 19.5 per cent of the state's population, 31.07 per cent of the state's physicians, and 19.84 per cent of the state's specialists. Of all physicians in cities of intermediate size, 28 per cent are classed as specialists and 72 per cent are in general practice.

In small communities (less than 5,000 population), 21.7 per cent of the state's population reside. Of the physicians practicing in these communities, only 8 per cent are specialists, leaving 92 per cent as general practitioners. It is rather surprising to know that there are 9 certified specialists practicing in these smaller communities.

TABLE XIX

DISTRIBUTION OF PHYSICIANS BY AGE AND SPECIALIZATION

Age Group Years	*Specialty* CERTIFIED	NON-CERTIFIED	GENERAL PRACTICE	TOTAL
Under 30	1	108	130	239
30-39	115	213	435	763
40-49	145	83	250	478
50-59	65	59	174	298
60-69	29	53	138	220
70-79	21	42	185	248
80 and over ..	2	12	80	94
TOTAL	378	570	1392	2340

Of course it is understood that medical services do overlap political boundary lines. This is especially true in the Kansas City area, as has previously been noted.

Kansas has three principal cities, two of which are

TABLE XX

AVERAGE AGE OF PHYSICIANS BY SPECIALTY

Status as to Limitation of Practice	*Average/or Mean Age in Years*
Specialty	
Certified	46.5
Non-Certified	42.8
General Practice	48.9
Average Age of all Physicians ..	47.0*

*Not an arithmetical average of above figures, but an average of the ages of all physicians in the state.

TABLE XXI

DISTRIBUTION OF PHYSICIANS BY SCHOOL OF GRADUATION AND BY SPECIALTY

Status as to Limitation of Practice	*Physicians* K. U. NO.	%	OTHER KANS. SCHOOLS NO.	%	OUT OF STATE NO.	%	TOTAL NO.	%
Specialty								
Certified	135	14.11	4	7.14	239	18.01	378	16.15
Non-Certified	180	18.81	9	16.07	381	28.7	570	24.36
General Practice	642	67.08	43	76.79	707	53.29	1392	59.49
TOTAL	957	100.00	56	100.00	1327	100.00	2340	100.00

TABLE XXII

DISTRIBUTION OF PHYSICIANS BY SCHOOL OF GRADUATION AS PERCENTAGE OF EACH SPECIALIZATION GROUP

School	*Specialty* CERTIFIED NO.	%	NON-CERTIFIED NO.	%	*General Practice* No.	%	*Total* No.	%
University of Kansas	135	35.7	180	31.6	642	46.12	957	40.90
Other Kansas Schools	4	1.0	9	1.6	43	3.09	56	2.39
Out of State Schools	239	63.3	381	66.8	707	50.79	1327	56.71
TOTAL	378	100.0	570	100.0	1392	100.00	2340	100.00

TABLE XXIII

DISTRIBUTION OF PHYSICIANS BY MAJOR GROUPS OF SPECIALTIES

Physicians	*Specialties* SURGICAL NO.	%	MEDICAL NO.	%	OTHER NO.	%	*Total* No.	%
Certified Specialists	173	40.71	139	35.73	66	49.25	378	39.87
Non-Certified Specialists	252	59.29	250	64.27	68	50.75	570	60.13
TOTAL	425	100.00	389	100.00	134	100.00	948	100.00
General Practice	1392							
Grand Total	2340							

considered as medical centers because they have hospitals of 200 beds or more for general use and because they furnish internships and residencies in two or more specialties. The rest of the state is pooled into a fourth group or category.

Looking at Table XVIII we see that Wichita has the largest number of specialized physicians both quantitatively and percentage wise, as compared with the other two cities. Kansas City has the fewest number of certified specialists. This is more apparent when one realizes that of the certified specialists in that city, most are located in the University of Kansas Medical Center. So Kansas City, Kansas, is a city of few certified specialists in private practice. People in that city often go across the state line to Kansas City, Missouri, when they are in need of the services of certified specialists because that is where more specialists are to be found.

It is commonly believed that specialization is particularly attractive to young men just finishing their medical education. Table XIX explores this possibility in Kansas. We have previously set up ages 30 to 59

years as the most effective age range in the practice of medicine. This age range includes 86.4 per cent of all certified specialists in Kansas, 62.2 per cent of all non-certified specialists, and 58.1 per cent of all general practitioners. From another point of view it is interesting to note that those physicians who are specialists (both certified and non-certified) make up 44.1 per cent of the 30-59 year age group.

Table XX gives the average age in the three categories as to specialization. Those practicing specialties are a little younger than those in general practice.

The University of Kansas School of Medicine emphasizes the training of general practitioners. Table XXI indicates the tendency of its graduates to specialize. Of the University of Kansas graduates now in the state, 67.08 per cent are in general practice, as compared with 14.11 per cent who are certified specialists and 18.81 per cent who are non-certified specialists.

A higher percentage (76.79) of graduates of other Kansas schools are in general practice, but this is to be expected as they received their training before specialization became emphasized as it is today.

Out of state graduates show 53.29 per cent as being in general practice as compared with 46.71 per cent who are specialists (both certified and non-certified). University of Kansas graduates have tended less toward specialization than have out of state graduates.

The data presented in Table XXI have been rearranged in Table XXII. This shows the proportionate distribution of each group of physicians (by school of graduation) to the ranks of specialists. All three groups make their largest contribution in the non-certified group of specialists as compared with those who are certified.

Let us now group the specialists in Kansas into three main categories: surgical, medical, and other, as shown in Table XXIII.

Of the 2,340 physicians in Kansas, 948 are considered as specialists; 60 per cent are non-certified. Of the specialists, 425, or 44.8 per cent, are in the field of surgery and its subdivisions; 389, or 41 per cent, are in medical specialties; and 134, or 14.2 per cent, are in all of the other specialties. Let us break the data down further to show the distribution of physicians by the field of specialty.

Table XXIV indicates the number of specialists in Kansas (certified and non-certified) in each field of specialty.

TABLE XXIV

DISTRIBUTION OF PHYSICIANS BY FIELD OF SPECIALTY

	Specialty		
Specialty	CERTIFIED	NON-CERTIFIED	*Total*
I. Surgical	*173*	*252*	*425*
1. Surgery	55	126	181
2. Proctology	1	1	2
3. Neurosurgery	4	4	8
4. Orthopedic Surgery	17	14	31
5. Plastic Surgery	1	4	5
6. Obstetrics	1	5	6
7. Gynecology	0	1	1
8. Obstetrics and Gynecology	22	32	54
9. Ophthalmology	26	13	39
10. Otorhinolaryngology	27	31	58
11. Ophthalmology plus Otorhinolaryngology	6	5	11
12. Urology	13	16	29
II. Medical	*139*	*250*	*389*
13. Dermatology	8	6	14
14. Internal Medicine	64	94	158
15. Allergy	0	3	3
16. Cardiovascular Diseases	0	1	1
17. Gastroenterology	0	0	0
18. Tuberculosis	0	2	2
19. Pediatrics	29	27	56
20. Psychiatry	6	102	108
21. Neurology	0	0	0
22. Psychiatry plus Neurology	32	15	47
III. Others	*66*	*68*	*134*
23. Anesthesiology	11	24	35
24. Pathology	16	15	31
25. Clinical Pathology	0	0	0
26. Bacteriology	0	0	0
27. Radiology	30	16	46
28. Industrial Practice	0	1	1
29. Others	9	12	21
TOTAL	378	570	948
General Practice			1392
GRAND TOTAL			2340

Summary

For the state as a whole, and out of the 2,340 resident physicians, 948, or 40.5 per cent, regard themselves as specialists. Six hundred and seventy-five of the specialists, or 71.2 per cent, are located in the three principal cities of the state. However, these cities only have 21.2 per cent of the state's population. In these same cities, 675, or 63.6 per cent of

(Continued on Page 326)

Cryptococcosis

Treatment of Two Cases with Stilbamidines

WIRT A. WARREN, M.D., G. GAYLE STEPHENS, M.D., *and* ERNEST W. CROW, M.D., *Wichita*

To 1951 the number of reported cases of systemic cryptococcosis approached 220.[2] Thirty-three of these patients were still alive to the publication dates. Ten more cases have been added as interest in the disease increases.[3] A nearly hopeless prognosis heightens the need for an effective therapeutic attack.

The aromatic diamidines, particularly stilbamidine, Propamidine, and diethylstilbesterol, show antimycotic effects.[1] In blastomycosis, both cutaneous and systemic, the use of stilbamidine has been gratifying.[4] A morphological likeness between this fungus and cryptococcus leads one to hope that stilbamidine might also be useful in treating cryptococcus infections. Miller et al. in 1952 reported using stilbamidine unsuccessfully in three patients with cryptococcosis.[12] In 1953 these authors reported like results in experimental infections in mice.[13] Sensitivity tests are not recorded.

Our two cases were similarly treated, the first mainly with stilbamidine, the second with a presumably less toxic derivative, 2 hydroxystilbamidine. In vitro sensitivity tests of these drugs on the organisms were obtained in each case.

Two cases of systemic cryptococcosis are reported which were treated unsuccessfully with stilbene derivatives. Case 1 received stilbamidine and negligible amounts of 2-hydroxystilbamidine. Case 2 received ONLY 2-hydroxystilbamidine. The in vitro sensitivity of these organisms to 2-hydroxystilbamidine covers a wide range of concentrations, but in Case 2 antimycotic activity is demonstrated.

Case 1

History (Obtained from wife): W. M., a 61-year-old white male, was admitted to Wesley Hospital on April 19, 1953. He had been acutely ill for five days with persistent frontal headache, fever of 100 to 103 degrees Fahrenheit, apathy, and drowsiness. On April 18 a tonic seizure occurred and the patient was unconscious for a short time. Following this seizure his speech was slurred, and he gradually became stuporous.

The past history was unimportant except for emotional instability and excessive fatigue for several years. He had been examined in a Veterans Administration hospital in 1948, but apparently no disease was found.

Physical examination: Temperature was 102 degrees Fahrenheit, pulse rate 80, respiratory rate 20, noisy but not labored.

The patient was emaciated, stuporous, and acutely and seriously ill. The pupil of the left eye was distorted by an old injury, but the right pupil reacted normally to light. The right fundus was normal, but the left fundus could not be seen. Cervical rigidity was marked. Ankle jerks were absent bilaterally. There was no Babinski sign, but an Oppenheim sign was found on the left. The remainder of the physical examination was normal.

Hospital course and treatment: The patient remained stuporous and gradually developed spasticity in the left upper and lower limbs. His temperature was of a septic type, spiking to 104 degrees Fahrenheit daily. Cryptococcosis was suspected from the initial spinal fluid examination and was confirmed on repeated examination. The organism was also recovered from gastric washings. A culture was sent to Washington University in St. Louis, and the organism was identified there also as Cryptococcus neoformans.

Initial treatment was supportive and included penicillin and streptomycin. Stilbamidine was started April 25 in daily doses of 450 mg. given intravenously in 1000 cc. of fluid. After four days the daily dose was reduced to 300 mgm. for three days, then to 150 mgm. for six days. On the 14th day 450 mgm. was given and on the 15th day 300 mgm. The total amount given was 4.35 Gm. A few doses of 2-hydroxystilbamidine were given during the last week of the patient's illness.

On the day after stilbamidine was begun the temperature leveled off at 100 to 102 degrees Fahrenheit. The spinal fluid cell count decreased, but organisms were still present. There was slight temporary improvement in the patient's mental status, and there

appeared to be some clearing of infiltrations found in chest x-rays. Later the patient became completely unresponsive and unable to move his lower extremities or to swallow. The temperature rose to 107 degrees Fahrenheit on May 9, 1953, and he died the next day. Autopsy was not permitted.

Significant laboratory findings: Initial white blood count 18,900; differential—segs 79 per cent, bands 5 per cent, lymphs 16 per cent.

Spinal fluid examination:

April 20, 1953—cells 241 per cu. mm., 60% polys, 40% lymphs

April 21, 1953—cells 396 per cu. mm., 60% polys, 40% lymphs

Pandy test	2+
Sugar	54 mgm. %
Total protein	184 mgm. %
Chloride	692 mgm. %
Colloidal gold	0122333100

April 22, 1953—cells 393 per cu. mm., 78% polys, 22% lymphs

April 23, 1953—cells 310 per cu. mm., 69% polys, 31% lymphs

April 25, 1953—cells 340 per cu. mm., 70% polys, 30% lymphs

April 28, 1953—cells 62 per cu. mm., 23% polys, 77% lymphs (3 days after starting stilbamidine)

Case 2

History: The patient was a 59-year-old white male admitted to Wesley Hospital on May 9, 1955, with a chief complaint of headache. He had been seriously disabled for 25 years with chronic tophaceous gouty arthritis; polycythemia vera had existed since 1946; and he also had chronic pyelonephritis with hydroureter and hydronephrosis. He had been taking cortisone 25 mgm. three times daily for two years and probenecid 0.5 Gm. twice daily for six months.

One week prior to admission the patient developed a severe frontal headache with occipital radiation. This was constant and steadily increased in intensity. The day of admission fever, slight confusion, and blurring of vision were noted.

Physical examination: Temperature was 100.4 degree Fahrenheit, pulse rate 120, respiratory rate 24, blood pressure 145/90.

The patient was confused and complained bitterly of headache. There were tophi on ears, elbows, knees, and fingers with accompanying joint deformity. Tendon reflexes were hyperactive but equal. There was no nuchal rigidity, and the remainder of the physical examination was normal.

Hospital course and treatment: On admission a diagnosis of encephalitis was considered. Spinal puncture showed an initial pressure of 300 mm. of water with a clear spinal fluid. The spinal puncture gave the patient much relief. The usual dosage of cortisone was continued, and aspirin was given for pain.

During the next five days there was sufficient symptomatic improvement to allow the patient to be up in his room. On May 15, 1955, he again became lethargic and developed diplopia. He remained thus for two weeks—the body temperature never being more than 100 degree Fahrenheit.

The second spinal fluid on May 17, 1955, showed a yeast-like organism which we thought was a contaminant. Culture of the third fluid on June 1, 1955, again demonstrated this organism which proved to be Cryptococcus neoformans. This culture was sent to the U. S. Public Health Laboratory in Kansas City, where the diagnosis was confirmed.

On June 1, 1955, the patient had a transient episode of unconsciousness. After the spinal tap this quickly disappeared. The spinal fluid pressure was then 340 mm. of water. On June 2, 1955, we started 2-hydroxystilbamidine in a daily dosage of 250 mgm. given intravenously in 500 cc. of fluid. This was continued until the patient's death on June 16, 1955. He was also given sulfadiazine 2.0 Gm. initially, followed by 1.0 Gm. every four hours for two days, then 2.0 Gm. every four hours. Six drops of saturated solution of potassium iodide were given three times daily.

No particular change in his condition was noted until June 9, 1955, at which time lethargy was marked, and there was weakness of the left lateral rectus and right facial muscles. A Babinski sign first appeared on the right and by June 15 was bilateral. Difficulty in swallowing was followed by coma, and he died on June 16, 1955.

Significant laboratory findings: May 9, 1955, blood urea nitrogen 57 mgm. per cent, serum uric acid 14.8 mgm. per cent.

Spinal fluid examination:

May 9, 1955—cells 450 per cu. mm., 51% polys, 49% lymphs

Pandy test	+
Sugar	57 mgm. %
Total protein	123 mgm. %
Chloride	687 mgm. %
Colloidal gold	0122221000

May 17, 1955—cells 111 per cu. mm., 50% polys, 50% lymphs

June 1, 1955—cells 114 per cu. mm., 55% polys, 45% lymphs

June 14, 1955—cells 3,567 per cu. mm.

Chest x-ray revealed a small annular shadow at the left base. This had been noted on a film in February 1955 but had not significantly changed.

At autopsy there were tubercle-like nodules on the dura and pia arachnoid over the brain and spinal

cord. These lesions contained cryptococci as did an area of abscess in the lung.

Discussion

Since the first recognition of cryptococcosis nearly 50 years ago, the search for a reliable treatment has been in vain.[16] Carton, in a very complete review, lists 34 chemicals and two physical methods which have been tried.[2] These include antiseptics, dyes, biologicals, heavy metals, vitamin D, thymol, and antibiotics. Some important experimental work was done by Kligman and Weidman in 1948.[9] In vitro studies were made with a great variety of agents which included fatty acids, sulfonamides, naphthaquinones, thiocarbamate derivatives, antibiotics, and certain members of the stilbene family. Appreciable inhibitory effects were observed from certain quinones, thiocarbamates, and antibiotics. Of the latter group actidione seemed to be almost specific for Cryptococcus neoformans. However, when this antibiotic (or any of the other agents) was given to mice, before or after intraperitoneal inoculation with the fungus, it invariably failed to prevent or overcome the infection.

Reports disagree concerning the effects of penicillin. Dawson et al. include the Cryptococcus in a list of organisms susceptible to penicillin;[5] Hamilton and Thompson report the case of a patient who died, but in whom they felt that penicillin partially reversed laboratory findings.[7] Others have found penicillin to fail completely.

Sulfadiazine has perhaps been used more in this disease than any other drug excepting iodides, but aside from the one evident cure of Marshall and Teeds, there have been no apparent beneficial effects.[8, 10]

Terramycin was used unsuccessfully in one case, while intrathecal tyrocidine produced a remission of at least nine months in another case.

Intravenous alcohol 5 per cent is said to have produced good results in one case; however, this was an atypical one, and we are aware of no follow-up.

Actidione (cycloheximide), an antibiotic produced by Streptomyces griseus, has been intensively investigated. Carton treated four cases with it; none has been cured, but two patients were alive and seemingly improved at the time of the report.[2] Wilson and Duryea treated a case with actidione and the symptoms were wholly arrested, and spinal fluid became sterile, and the patient was well 20 months later.[17]

Because of the antimycotic activity of stilbamidine and its clinical usefulness in blastomycosis, we chose to try it (and later the 2-hydroxy derivative) in the treatment of our two cases of systemic cryptococcosis. The in vitro sensitivity studies on the organism isolated in each of these cases is as follows:

Case 1	*Agent*	*Case 2*
Inhibition of growth at 1000 mcg. per cc.	Stilbamidine	Not tested
No inhibition up to 10,000 mcg. per cc.	2-hydroxystilbamidine	Inhibition of growth at 20 mcg. per cc.
Resistant	Penicillin	Resistant
Resistant	Streptomycin	Resistant
Resistant	Aureomycin	Resistant
Resistant	Chloromycetin	Resistant
Resistant	Terramycin	Resistant
Resistant	Gantrisin	Resistant
Resistant	Triple sulfas	Resistant
Resistant	Sulfamerazine	Resistant
Resistant	Polymixin B	Resistant
Resistant	Bacitracin	Resistant
Not tested	Erythromycin	Resistant
Not tested	Furadantin	Resistant
Not tested	Neomycin	Resistant
Not tested	Achromycin	Resistant
Somewhat sensitive	Sulfadiazine	Resistant
Somewhat sensitive	Sulfathiazole	Resistant

From the sensitivity tests we conclude that a clinical response to stilbamidine or 2-hydroxystilbamidine in Case 1 would be highly unlikely, yet a slight transient improvement *did* occur; whereas our studies in Case 2 allowed hope for benefit, since a concentration of 20 mcg./cc. is attainable in body fluids; nonetheless this patient showed no clinical response. (Of some interest here is the fact that Case 2 got large doses of sulfadiazine in addition to 2-hydroxystilbamidine.)

The fairly good degree of sensitivity shown by the organism of Case 2 in the *in vitro tests* suggests that 2-hydroxystilbamidine has antimycotic action and merits further study. Its use in another deep mycocis, coccidioidomycosis, has recently been reported by Snapper et al.[14]

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(Continued on Page 326)

Abdominal Pregnancy

Report of Successful Management of One Case

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The following report of a rare surgical entity is presented in the hope that it will be of interest and may be of benefit to physicians who have not encountered the problem of managing a case of abdominal pregnancy.

The patient, a 42-year-old nulliparous white female, was first seen by us on June 15, 1953. Her chief complaint was intermittent and cramping pain in the left lower quadrant of the abdomen. She had first had the pain two weeks previously. She reported her most recent menstrual periods as having begun on April 5, 1953, and on May 1, 1953.

A physician she saw on June 1, 1953, related her symptoms to the beginning of the menopause and prescribed oral estrogen as symptomatic relief. After taking four or five doses of the medication, she became nauseated and developed severe anorexia, pain, and cramping in the left lower quadrant, progressively severe.

The patient reported to us that she "had never been able to get pregnant," in spite of the fact that no contraceptive devices had been used. She gave no history of gynecological infection and felt that she had always been in good health. Two years previously she had undergone surgery for removal of a mole on the left lower leg. She reported that the mole was diagnosed as malignant. The family history was noncontributory.

Physical Examination

The patient was well developed, well nourished, and appeared to be the stated age. Her temperature was 99.6 degrees, and blood pressure was 110/70. Mucous membranes and skin appeared pale and sallow. Her attitude was that of a depressed person, but she did not appear to be acutely ill.

Palpation of the abdomen revealed a mass in the left lower quadrant approximately 6 by 6 cm. It was fixed and soft, and the entire quadrant was tender to palpation.

On pelvic examination the vaginal vault was found to be filled with a profuse, foul smelling, dirty gray discharge. The cervical os was small and round with some erosion and was apparently the source of the discharge. The cervix itself was innocent in appearance. The uterus was about twice normal size and seemed softer to palpation than normal. The right adnexa was free but tender, and the left adnexa was examined with difficulty because of extreme tenderness. It was the examiner's impression that the mass was adjacent to the left tubo-ovarian region.

On vaginal-rectal-abdominal examination the uterus, although enlarged, could be distinguished from the mass in the left lower quadrant.

Laboratory examination revealed hemoglobin 59 per cent; red blood count 3,440,000; white blood count 8,000; Wassermann negative, and urinalysis negative.

Experience with one patient leads to the conclusion that a physician should suspect pregnancy in any female of childbearing age with abnormal bleeding, anticipate hemorrhage in the disruption of an abdominal pregnancy, and avoid removal of the placenta from its attachment if hemostasis is secure.

Pregnancy was considered, but a conservative regimen was chosen for two reasons: (1) The patient had never before conceived; (2) The symptoms of fever and profuse vaginal discharge pointed strongly to an inflammatory or malignant mass in the left tubo-ovarian region. In addition, she was anemic and in need of physical rehabilitation before surgery.

She was given antibiotics, B_{12}, and iron. After surprising improvement on those medications, she was admitted to the hospital for surgery on July 7, 1953. Examination at that time showed that the mass had almost doubled in size, making surgery imperative.

Urinalysis on the morning of surgery revealed a two plus qualitative Benedict's test, but a fasting blood sugar revealed 110 mgm. per cent of glucose.

Surgery

The abdominal cavity was entered through a 10 cm. lower midline incision. A fixed, soft membranous mass containing fluid was found in the left lower quadrant. It was attached to the lateral wall of the abdominal cavity and adherent to the mesentery and small bowel posteriorly, the cul-de-sac and sigmoid inferiorly, and to the broad ligament and the posterior

portion of the uterine fundus anteriorly. The mass measured approximately 10 by 10 cm.

On palpation of the mass, the uterus was antiflexed. This ruptured the cystic mass and brought forth an almost exsanguinating hemorrhage, difficult to control because of a bloody field and friable tissues. To combat shock, 1,000 cc. of whole blood was started.

A fetus measuring 14 cm. was taken from inside the amniotic sac, the cord was ligated and severed, and loose fragments of placental tissue were removed. Ligatures were useless in this friable mass of placental tissue. A large curved hemostat, which apparently contained the base of the implantation on the lateral wall of the abdomen, was left in place. Gelfoam packs were placed in the cul-de-sac, and a large pack was loosely placed posterior to the uterus, allowing about one-half of the pack to protrude, along with the hemostat, through the distal part of the abdominal incision. When hemostasis was secure, the abdominal incision was closed in the usual manner.

Postoperative Course

The patient was returned to her room in fair condition. Blood volume was replaced, and she was given antibiotics and fluids. These, with general supportive measures, contributed to an uneventful postoperative course. The pack was removed on the third postoperative day, and the hemostat was removed on the fourth postoperative day.

A fecal fistula developed on the 11th postoperative day. Neomycin therapy was instituted, and four days later the fistula closed spontaneously.

The patient was discharged on her 20th hospital day. She was ambulatory, and her abdominal incision was well healed except for the lower part which required dressings for the following three weeks. She enjoyed complete recovery.

Conclusion

Our experience with this patient leads us to conclude that a physician should (1) suspect pregnancy in any female of child bearing age with abnormal uterine bleeding; (2) anticipate hemorrhage in the disruption of an abdominal pregnancy; (3) avoid removal of the placenta from its attachment if hemostasis is secure.

Abdominal pregnancy is a rare entity occurring once in about 12,500 pregnancies, 3 to 1 times more often in Negroes than in white women. The pathogenesis is apparently that of tubal pregnancy precipitated by an underlying chronic salpingitis. As in this case, the highest incidence is in primigravidae who may or may not give a history of pelvic inflammatory disease.

Perhaps the tubal pregnancy is aborted, or partially aborted, to attach itself to any organ or viscus in the abdomen. It is apparent that some of the choronic villi must retain their integrity while the secondary site is being invaded. The tubal rupture is thought to occur between the third and fourth month of gestation.

Lower abdominal pain is an outstanding symptom. Other symptoms are nausea, vomiting, syncope, and shock, depending on the amount of bleeding that occurs at the time of rupture. Symptoms, except for lack of menstruation, may be absent for a time. At about five months gestation, the patient develops painful fetal movements, and it is during this time that fetal bones can be outlined by x-ray for study of the position of the fetus. It is interesting to note that the uterus will be enlarged to about three times its normal size and show decidual reaction.

After a positive diagnosis has been reached, there is no reason for delaying surgery. Spontaneous rupture of the sac may result in an exsanguinating maternal hemorrhage.

There is apparently no one method of managing the placenta that is adaptable to every situation the surgeon may encounter. However, since there is no apparent danger in leaving the placenta intact, that may be done.

It is important to conserve maternal blood and to have at least 1,000 cc. of compatible blood available for transfusion.

Closing the abdomen with or without drainage is controversial and must be left to the surgeon at the time of operation.

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In the past eight years, the proportion of the nation's total medical expenses met by voluntary health insurance has increased three times as fast as the proportion of Americans holding such insurance, according to Health Information Foundation.

PRESIDENT'S PAGE

DEAR DOCTOR:

Truly, it would be presumptuous to assume that momentous achievements shall be accomplished in the short period of one year.

A gifted surgeon-teacher once said, "Your sutures are merely a hint to nature, suggesting where to start the healing process." So, it in no manner disregards the high honor, nor belittles the responsibilities nor the importance of his office, when one confesses he can only hope to "hint" at important activities for his organization.

For this 99th year of the Kansas Medical Society there are many tasks to challenge our mettle.

—*Item:* That product of years of "blood, sweat and tears," the amazing final accomplishment of a sound medical practice act and basic science law. Unbelievably, they are fact and in force. They are yet to be implemented, put into gear, and set into operation.

—*Item:* The great responsibility of the medical profession to take positive action to help curtail the slaughter on the highways that takes a human life every 14 minutes, produces a serious injury every 24 seconds, 24 hours a day.

—*Item:* The continuing responsibility to expand and develop Blue Shield, to further aid our patients to meet the difficult economic problems of illness; to further enlighten and familiarize our membership with the philosophy, aims, and tremendous value of Blue Shield to the American public and, therefore, also and incidentally, to ourselves.

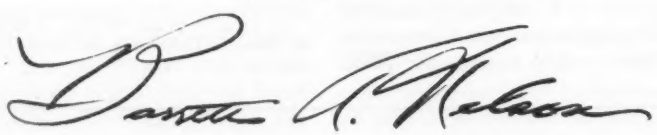
—*Item:* The tremendous need for psychiatric services which can be available to our patients and which can be obtainable within their financial capacities. Coupled with this, the great need for psychiatric orientation and understanding on the part of the profession as a whole, as well as closer liaison with, and a better understanding of, the problems of the rest of the profession, on the part of the doctor who specializes in psychiatry.

—*Item:* The problem of emphasizing to the profession in Kansas the vital importance of the American Medical Education Foundation and the urgent need for our active support.

—*Item:* The day by day operation of one of the most vigorous, active, individualistic, and aggressive of all the state medical societies.

Not to mention the long list of many and various tasks that are pressing for the attention of the conscientious energetic committees which are the real, functioning, productive machinery of our organization.

Humbly yours,



President

EDITORIAL COMMENT

Uniform Narcotics Act

On July 1, 1957, Kansas will have in effect a new act controlling narcotic drugs. This is in compliance with the recommended federal act and is known as the Uniform Narcotic Drug Act. It is a rather long bill, carefully regulating a great many possible situations of which this summary will consider only those of interest to medicine. The bill also regulates use of narcotics by the dentist, the veterinarian, the manufacturer, the wholesaler, the pharmacist, etc.

" 'Physician' shall mean any person authorized by law to treat sick and injured human beings in this state and to use narcotic drugs in connection with such treatment." The law makes no change in who is licensed to use narcotics. Everyone currently licensed to prescribe narcotics may do so in the future. No new privileges are granted.

" 'Narcotic drugs' means coca leaves, opium, cannabis, isonipecaine, amidone, isoamidone, ketobemidone and every other substance neither chemically nor physically distinguishable from them; any other drugs to which the federal narcotic laws may now apply; and any drug found by the state board of pharmacy and the state board of health, after reasonable notice and opportunity for hearing, to have an addiction-forming or addiction-sustaining liability similar to morphine or cocaine, from the effective date of determination of such finding by said state board of pharmacy and state board of health."

It will be noted that anything that the federal narcotic laws list as narcotics or whatever the state board of pharmacy and the state board of health list as narcotic drugs will come under this act.

The act states that a pharmacist in good faith may sell and dispense narcotic drugs to any person upon a written prescription of a physician, dentist, or veterinarian, dated and signed on the day issued and bearing the full name and address of the patient for whom the drug is dispensed, and the full name, address, and registry number under the federal narcotic laws of the person prescribing. The person filling the prescription shall write the date of filling and his own signature on the face of the prescription. The prescription shall be retained on file by the proprietor of the pharmacy in which it is filled for a period of two years, so as to be readily accessible for inspection by any public officer or employee engaged in the enforcement of this act. The prescription shall not be refilled.

The act then says that such narcotic drugs possessing little or no addiction liability and which are so listed by the federal laws may be dispensed upon an oral prescription.

Even then a pharmacist ". . . may sell to a physician, dentist, or veterinarian, in quantities not exceeding one ounce at any one time, aqueous or oleaginous solutions of which the content of narcotic drugs does not exceed a proportion greater than 20 per cent of the complete solution, to be used for medical purposes. A physician or a dentist, in good faith and in the course of his professional practice only, may prescribe, administer, and dispense narcotic drugs, or he may cause the same to be administered by a nurse or interne under his direction and supervision."

Exempted is the selling, or dispensing at retail, of any medicinal preparations that contain in one fluid ounce, or if a solid or semi-solid preparation, in one avoirdupois ounce, not more than one grain of codeine or of any of its salts, or not more than one-sixth grain of dihydrocodeinone or any of its salts, provided that the preparation shall contain, in addition to the narcotic drugs, some other drug conferring upon it medicinal qualities and provided that it is used in good faith as a medicine. Every physician must keep a record of narcotic drugs received by him and a record of all such drugs administered, dispensed, or professionally used by him other than by prescription.

An exception is that ". . . no record need be kept of narcotic drugs administered, dispensed, or professionally used in the treatment of any one patient, when the amount administered, dispensed, or professionally used for that purpose does not exceed in any 48 consecutive hours (a) four grains of opium, or (b) one-half of a grain of morphine or of any of its salts, or (c) two grains of codeine or of any of its salts, or (d) one-fourth of a grain of heroin or of any of its salts, or (e) a quantity of any other narcotic drug or any combination of narcotic drugs that does not exceed in pharmacologic potency any one of the drugs named above in the quantity stated."

The patient may lawfully possess narcotic drugs only in the containers in which they are delivered to him by the person selling or dispensing the same.

All narcotic drugs which are not lawfully owned shall be forfeited and disposed of by the secretary of the state board of health.

On the conviction of any person in violation of this act, the licensing board under which he practices shall be ordered to revoke his license, which may be reinstated only upon proper showing and for good cause.

The law states that information communicated to a physician in an effort unlawfully to procure a narcotic drug, or unlawfully to procure the administration of any such drug, shall not be deemed a privi-

leged communication. It then sets a number of penalties for violations and states that cities of the first, second, and third classes may adopt ordinances, not in conflict with this act, for the purpose of controlling traffic in narcotic drugs within their corporate limits.

Also becoming a law on July 1, 1957, is a new act controlling ". . . (a) the salts and derivatives of barbituric acid or compounds or preparations or mixtures thereof; (b) amphetamine, its salts and derivatives or compounds, preparations or mixtures thereof; and (c) other drugs or compounds, preparations or mixtures thereof which the state board of health shall find and declare by rule or regulation duly promulgated after reasonable public notice and opportunity for hearing to have a dangerous hypnotic, somnifacient or stimulating effect on the body of a human or animal; except that the term 'drug' shall not include any drug the manufacture or delivery of which is regulated by the narcotic drug laws of this state." As used in this act a "dangerous drug" means one that is unsafe for use except under the supervision of a practitioner because of its toxicity or other potentiality for harmful effect, method of use, or collateral measures necessary to use. The words "somnifacient" and "stimulating" as used in this act shall have the meaning attributable in standard medical lexicons.

This act authorizes the board of health to promulgate necessary regulations for the administration and enforcement of this act. It provides for penalties for violations, requires records of purchases to be kept for two years, and declares that a telephone prescription "shall be promptly reduced to writing . . ." and that it may not be refilled "unless such refilling is specifically authorized by the prescriber."

The term "practitioner" is defined as "any person authorized by law to prescribe and administer drugs, as herein defined, in the course of his professional practice; professional practice of a practitioner means treatment of patients under a bona fide practitioner-patient relationship." Therefore, as in the previously described act, no new practice privileges are conferred.

Soviet Medicine

In a recent issue of *Problems of Communism,* a magazine published by the U. S. Information Agency, there is an article entitled "The Soviet Doctor's Dilemma" by Mark G. Field, an American sociologist who is with the Russian Research Center at Harvard. The article states that the Ministry of Health has established that a physician must see not less than six patients an hour and spend not more than ten minutes with each patient. Surgeons examine ten patients per hour, obstetricians six, otolaryngologists eight, and dentists two. One house call is considered the equivalent of three office visits.

Even this amount of time is not entirely spent with the patient. Of the ten minutes allowed for each patient, a period of eight minutes is required for the necessary paper work, and something under two minutes is devoted to listening to a description of symptoms and the examination.

Mr. Field visited the U.S.S.R. last summer in company with Paul Dudley White, M.D., of Boston. In this report Mr. Field says the ordinary patient puts up with a great deal of bureaucratic inefficiency and indifference and with all kinds of deficiencies and shortages. The emphasis is all on keeping the working man at his job, so physicians are forced to be more like policemen than like doctors, and they work in overcrowded facilities and often lack the most common medicines. However, even at this, medical services have improved in the last few years. By the end of 1955 there were 1,290,000 hospital beds and 334,000 doctors in Russia.

Mr. Field says, "The Soviet hero is not the healer but the man who builds machinery for industry or the one who 'fights' for the cause. By the same token, the financial rewards which doctors can command generally compare unfavorably with those of government officials or skilled industrial workers. At the Kharkov Electro-Mechanical Plant I was told that most qualified workers earned approximately 2,000 rubles a month. The average physician's salary was reported to be about 750 rubles to 800 rubles a month.

"A doctor is also at a relative disadvantage when it comes to obtaining housing and furnishings. Housing, for example, is assigned first to party and other officials, then to technical and other specialists. Only what is left over is available to physicians.

"It is interesting to note that in the Soviet Union medicine has become a woman's occupation. About seven to eight out of ten doctors are women."

Heedless Horsepower

In the year 1956, the Age of the Automobile came into its own. A multibillion dollar highway development program was passed by Congress, assuring the motoring public of wider, longer, straighter roads in the years to come. Automobile manufacturers fashioned dreams of steel; powerful, sleek, multicolored models with push-botton operation from dashboard to tail-light. And, the nation's drivers made certain that accident statistics came of age, too.

They killed, maimed, crippled and destroyed more men, women, children and property than ever before. While we began to blueprint America's vast

highway network of the future, heedless horsepower ran rampant on the roads of the present . . . warming up for the greater days that lie ahead.

From our statistical vantage point, we can see at a single glance every one of the accidents that occurred on our highways in 1956. We can see cars spinning out of control on wet or icy roads, pedestrians struck down as they cross against the light, weary, drunk, or inconsiderate drivers, gambling their reflexes against their lives . . . or the lives of others. We see the reckless young, dashing to a date with eternity; the careless old, spinning to disaster in an automobile that hasn't been checked in years. State by state, day by day, we can call off the grisly roll: 40,000 deaths, almost 6 per cent more than 1955; 2,368,000 injuries, up almost 10 per cent over 1955. One in every 70 Americans a statistic . . . a pain-wracked survivor, or a name in the obituary column.

But as we survey this appalling panorama, one pattern immediately stands out. It is a straight road on a clear day. Conditions perfect. A car that responds instantly to the driver's touch. One after another they zoom by; unthinking, unreflecting, letting heedless horsepower rush them to the unexpected accident that waits just down the road.

Heedless horsepower. It is a chronic disease of the Age of the Automobile. Its symptoms are many and various. The heavy foot on the accelerator; the eye fixed on the climbing speedometer; the hand on the horn; the mind idling while the car is in high. This is what we see in the vast majority of cases, as the grim spectacle of last year's accidents unfolds.

Over the past 30 years, many attempts have been made to embody safety in motor vehicle design and equipment. There can be no doubt that all steel bodies, safety glass, seat belts, padded instrument panels, power brakes have made automobiles less lethal to their occupants. However, all these safety devices are nullified by any combination of speed plus carelessness, thoughtlessness and lack of consideration.

The disease of heedless horsepower is highly contagious. It can be spread by an irresponsible word, an inflated claim, a careless example. And everyone who is in a position to influence drivers should learn that horsepower, in the hands of the heedless, is the fundamental cause of our ever-mounting toll of disaster.

However, it is the driver, not the manufacturer, the advertiser, or the salesman who must bear the greatest weight of blame. For it is the driver who can control the horsepower and use it safely for his greater ease and convenience. It is the driver who is lectured to, legislated at, prayed for, preached to . . . in every medium of public expression known to man. And it is the driver who nods sagely, promises readily, and forgets everything but his sense of overwhelming power when he steps on the gas.

Now we stand looking down on the widespread montage of the American highway of 1957. Already, heedless horsepower has taken its toll. But every year we get another chance to learn by experience, to profit from the deadly mistakes of the past. Slow down. We want to write to you . . . not about you next year.—*The Travelers Insurance Companies, Hartford, Connecticut*

Radiological Society Elects

A meeting of the Kansas Radiological Society was held in Kansas City in February, and the following officers were elected for the year: president, Dr. G. Sherman Ripley, Jr., Salina; vice-president, Dr. Louis G. Allen, Kansas City; secretary, Dr. James R. Stark, Wichita; councilor to the American College of Radiology, Dr. Charles M. White, Wichita; alternate councilor, Dr. A. M. Cherner, Hays.

Research in Cancer Chemotherapy

Cooperative studies in human cancer chemotherapy were begun recently by investigators at the University of Kansas School of Medicine and personnel of eight other teaching institutions. Initial studies will be on chronic leukemias and selected types of malignant growths. The work in Kansas will be done under a grant of $106,639 recently provided by the National Institutes of Health.

Workers on the campus in Kansas City will be Dr. Sloan Wilson, associate professor of medicine and oncology and head of the hematology section, and Dr. William E. Larsen and Dr. John Christianson, associates in the department of medicine.

Plans for the work were outlined at meetings held recently in New Orleans, Atlanta, and Miami. It is felt that the cooperative studies will develop more data in six months than could be developed in several years if each school were working independently. There are approximately 10,000 compounds to be tested in animal experiments before they can be tried on human subjects.

Working with the Kansas school on the project are the University of Mississippi, the Medical College of Alabama, the University of North Carolina, the Medical College of Virginia, the University of Miami, Washington University, Duke University, and Emory University.

Of drivers in 1956 highway accidents, 96.7 per cent had more than one year's driving experience.

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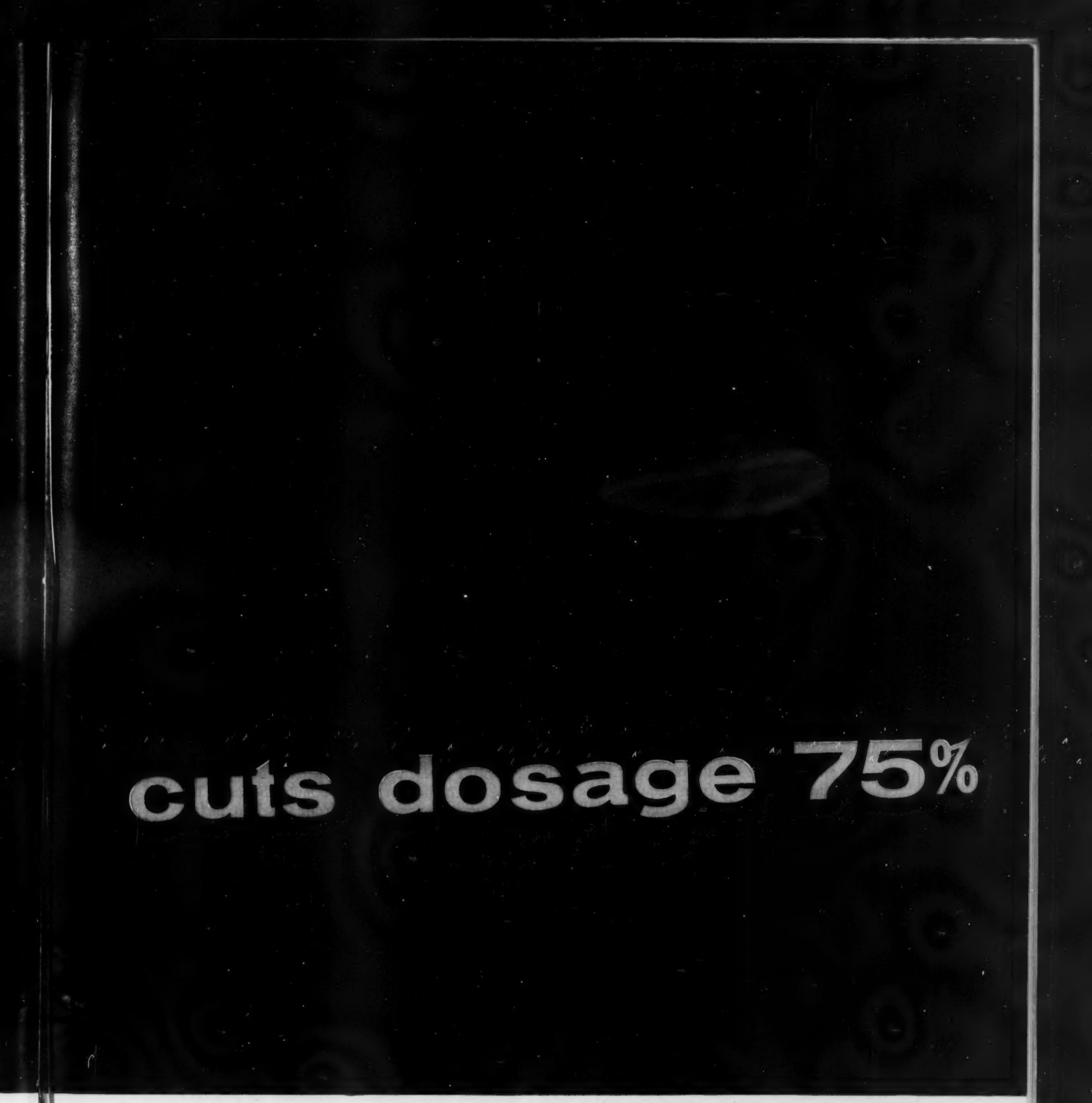

LOW DOSAGE: a total maintenance dose of only 2 tablets daily.

SOLUBILITY: prompt absorption, ready diffusion into body fluid and tissue.

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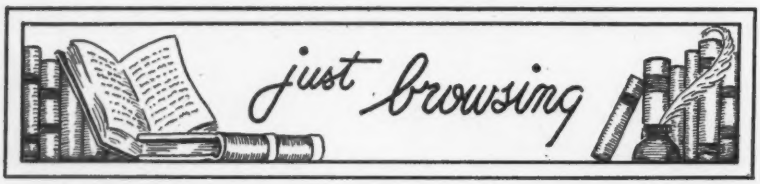

"Nature produces diseases and their antidotes. The pharmacist prepares and compounds, but the physician alone interprets symptoms, seeks out their causes and devises the cure. Unless sure of his remedy, it is as criminal to give it to a beggar as to a prince. . . .

"That wisdom is better than rubies has gone unchallenged for 30 centuries, until today the proverb is doubly true, for the wisdom of the present includes more perfect knowledge of the causes and control of diseases than was attained in any age of all the past. As a correct use of the facts now known to science would prevent most cases of sickness, and annually save millions of lives, how important that the masses be taught until everywhere the incantations of the voodoo shall give way to germicides, the darkness of superstition to the light of biology flooding the world.

"To remain in the front rank of the world's benefactors, the physician, too, must add to the sum of useful knowledge in terms that can be understood. 'All that a man hath will he give for his life,' and he should no longer be barred from knowing himself, from acquiring the best possible knowledge of physiology and hygiene, and from learning to outwit and destroy the micro-organisms that would make of him their prey.

". . . The doctor has stood aloof from the people too long, to their anxious inquiries has replied in an unknown tongue, and mystified what should have been made plain, until it is not wholly his own fault that the average individual knows less of himself than of almost everything else. You may doubt this statement, or say that it is not true, but the trainloads of patent dopes, the worse than worthless nostrums marketed annually, and the golden harvests gathered all the year by advertising charlatans, prove that it is true; . . .

"Education is the only remedy. Encourage study. Disseminate knowledge. Let all be informed upon sanitation and the nature of the more common maladies, and there will come ability to discern between quackery and skill, between the false and the true. And the physician's usefulness will keep pace, always, with the advance. Indeed, it is only where there is this intelligence that he can have the unswerving confidence of the patient and his friends, that cooperation in emergencies, so essential to the highest success in long and desperate combats with disease.

"A strictly scientific classification has not been attempted. As far as possible, plain, simple language has been used, and technical terms avoided. It is not expected that anyone, not possessed of special knowledge and experience, will attempt to treat the more serious forms of disease, but that the information here given will prove most valuable in preventing sickness, in accidents and other emergencies, and by enabling all to better cooperate with the physician in his best efforts to save life. If the work adequately meets, as we trust it will, the needs of those for whom it was written, we shall not consider that our long and laborious task has been in vain."

We are apt to think that the present-day dissemination of medical and scientific facts to the public through the medium of books, magazines and addresses is a new development; that we are exploring a new path in public relations for medicine. Such, however, is not quite the case. The foregoing quotations are from the introduction to a treatise* of over 800 pages, written for the general public by eleven physicians** and one dentist† who were members of the faculty of the old Kansas Medical College—the medical school of Washburn College, in Topeka. Under the editorship of W. E. McVey (who was also editor of the JOURNAL and secretary of the Kansas Medical Society at the time), the book was published by Herbert S. Reed, of Topeka, in 1900. It bears testimony to the fact that these faculty members were not unmindful of a responsibility in addition to that of treating patients and teaching medical students, and they devoted a considerable effort—the "long and laborious task"—toward fulfillment of that obligation. It makes interesting reading.—*O.R.C.*

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* The Human Machine: Its Care and Repair.

** Ida C. Barnes, Louis C. Duncan, B. D. Eastman, Joseph T. Lovewell, Robert S. Magee, John C. McClintock, Richard E. McVey, William E. McVey, John E. Minney, Theodore W. Peers, W. L. Schenck.

† Alfred C. Sloan.

LETTERS TO THE EDITOR

The following communication, written by L. E. Krause, D.S.C., Great Bend, concerns a subject thought to be of interest to JOURNAL *readers, the practice of chiropody today.*

Too many people, unfortunately, are misinformed about chiropody and its scope of practice. Most physicians probably know less about chiropody than any other branch of medicine. Many have difficulty in pronouncing the name correctly, perhaps because the first two syllables of the word sound as if "practor" were going to follow. Gould's Medical Dictionary defines a chiropodist (podiatrist) as a specialist in diseases of the feet. Kansas statutes relating to the practice of chiropody define a chiropodist (podiatrist) as a physician of the foot.

The curriculum of a modern chiropodist is similar to that of a dentist or a doctor of medicine. Chiropody does not use or teach cultist methods. It is a definite unit of medically trained students with known obligations toward the parent body of medicine, as well as a cognizance of a specialty practice. Our present education consists of a one- or two-year pre-medical course, followed by four years in one of six schools of chiropody and foot surgery recognized by the National Association of Chiropodists. The curriculum consists of about 4,400 hours or more in classrooms, laboratories, and clinics and includes bacteriology, pathology, histology, bio-chemistry, neurology, dermatology, orthopedics, anatomy, physiology, materia medica, pharmacology, surgery, physical diagnosis, and clinical and didactic chiropody. In addition, several states require a year of internship in a foot clinic or hospital after graduation before an examination for license will be given. All schools grant a Doctor of Surgical Chiropody degree (D.S.C.) except one which grants the degree of Doctor of Podiatry (Pod.D.). The degrees are synonymous.

In Kansas, the chiropody examining board is composed of two members of the medical examining board and one chiropodist. The examination is given at the same time and place as the medical examination. Here we have the unique position of being the only allied medical profession completely and wholly under the jurisdiction of the parent body.

Some chiropody schools are associated with universities so that, in many instances, leading members of the faculty teach at both medical and chiropody schools. Each school has a large foot clinic attached to it where students receive practical clinical instruction and experience.

Many of the nation's leading hospitals and clinics have active chiropody services, and others are constantly being added. Chiropodists are on the staffs of about 1,000 hospitals throughout the country. The medical departments of the armed forces have created commissions for our professional services.

Charles Mayo[1] put the problem succinctly in these words: "I am convinced that doctors of medicine, myself included, have paid too little attention to the feet in their relationship to the condition of the patient, and have made too cursory an examination of the feet considering their importance to people, with the beating they take and their potentiality as a source of comfort or discomfort. The doctor of medicine should be capable of recognizing foot ailments. When care and treatment of such conditions are necessary, he should refer the patients to those accredited and skilled in the specialty when such consultation is available."

Ruben Friedman,[2] clinical professor of dermatology, Temple University School of Medicine, put it another way. "It has been estimated," he said, "that disorders of the feet occur in 80 per cent of women and in 60 per cent of men. Few practitioners of the healing arts are as familiar with the almost infinite number and variety as well as the significance of the symptoms and diseases of the pedal extremities as the chiropodist. Once he is engaged in active practice, the chiropodist sees more patients suffering from one form or another of diseases of the feet than does any practitioner of medicine."

Poindexter[3] states, "In the field of chiropody, the practitioner is invaluable to the generalist, internist, or pediatrician treating such diseases as diabetes mellitus, cardiovascular diseases and arthritis. The orthopedist, neurologist, and surgeon have need for his services. Chiropody is more of medicine in a limited field, and this is all the more reason for an equivalent educational background. As a member of the Idaho Board of Examiners in Chiropody, as well as chairman of the State Board of Medicine, I have had the opportunity to write questions for your examinations and to correct them. The caliber of the papers is exceptionally good, and from them any physician could learn of your background, but it is difficult to communicate this in a satisfactory way."

Editorial comments in the *Canadian Medical Association Journal*[4] stated: "Morton, who can speak with authority, holds that 'The foot is the only part of the body for which prevailing ideas of care and treatment have remained practically the same for 40 years.' Signs are not wanting, however, to show that the lag of which Morton complained is being gradually overcome. More and more textbooks are dealing with the feet, and chiropody is slowly but surely establishing itself. The chiropodist is no longer only one who extracts corns, useful as that may be. He

is now a trained professional man with professional and ethical standards like our own and based on rigid training. He specializes as does the dentist, on a limited field of the body outside of which he does not venture."

Those of us who specialize in the feet feel that we deal with, as Morton described, the most neglected phase of medicine today. Some of us suspect that much of the stress and strain of modern living, as emphasized by Selye and his workers, can possibly be traced to the use and abuse of this organ. For those physicians who might doubt this, I would suggest you examine the feet of all patients as carefully as you would the liver, lungs, heart, etc. Many of your patients suffering fatigue, leg-back ache, nervousness, may present something as simple as a deep plantar heloma. If you question the significance of this finding, you yourself might try walking a few days with a stone taped to the plantar of the great toe joint. We sincerely believe the old axiom, "When your feet hurt, you hurt all over," has greater significance than we dared think.

We would like for you to believe the modern chiropodist fills a definite need in the medical-orthopedic fields and is well fitted to assume the responsibilities of caring for the nation's feet. It is not suggested that all current practitioners of chiropody are necessarily competent or modern, but I urge the discerning physician to evaluate the chiropodist in his community and, whenever possible, employ his services in promoting the foot health of the American people. Chiropody needs, seeks and hopes to earn the cooperation of medicine.

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1. Mayo, Charles: Journal of National Association of Chiropodists 41:35 (June) 1951.
2. Friedman, Ruben: Delaware State Journal of Medicine 25:127 (June) 1953.
3. Poindexter, S. M.: Journal of National Association of Chiropodists 46:10 (Oct.) 1956.
4. Editorial Comment: Canadian Medical Association Journal (Sept.) 1953.

BLUE SHIELD

"A strong Blue Shield is vital to the freedom of medical practice, and an understanding physician is vital to Blue Shield." This was the underlying theme of a highly successful professional relations conference held by the national association of Blue Shield Medical Care Plans in Chicago, February 11-13.

Some 110 Blue Shield professional relations directors and staff members were joined by more than 70 physician-trustees of local Blue Shield plans and 35 executive secretaries of sponsoring county and state medical societies. The conference was conducted by the national Blue Shield Professional Relations Committee, whose chairman is Dr. Fredrick H. Good of Denver, president of the Colorado Blue Shield Plan.

Keynoter of the conference was Dr. Robert L. Novy, of Detroit, national president of Blue Shield Medical Care Plans, who emphasized that the ideals and purposes of Blue Shield are precisely the same as the age-old ideals and purposes of medicine: to serve people singlemindedly, regardless of personal profit.

"Blue Shield safeguards the basic freedoms of medical practice which are fundamental to good medical care," Dr. Novy said. "Blue Shield hopes to strengthen the doctor's traditional way of practicing medicine, not to change it or destroy it. Blue Shield protects the patient's right to choose his doctor, the doctor's right to accept or reject the patient, and their common right to an inviolate confidential relationship."

Dr. Novy pointed out that in the 15 years since Blue Shield was created, a whole new generation of doctors has come into practice who know nothing of the struggle and sacrifice of its founders. Many of these doctors take Blue Shield for granted, and the success of Blue Shield has even led many of their older colleagues to take it for granted, too.

Indifference, apathy, and complacence can be fatal to Blue Shield and to the whole voluntary medical care prepayment program. "Blue Shield deserves the doctor's whole-hearted support because it is fashioned in the doctor's own image; it is his own creation; and it is designed to strengthen the freedoms that he and his patients want to keep strong and safe," Dr. Novy concluded.

Dr. Francis Collins, Topeka, president of Kansas Blue Shield, Dr. E. Burke Scagnelli, Dodge City, and Dr. James B. Fisher, Wichita, were representatives of the Kansas Blue Shield board at the Chicago conference. Other Kansans attending included Rueben Dalbec of the Kansas Medical Society staff, Proctor Redd, director of public and professional relations of Kansas Blue Shield, and Tom Reed, manager of physician relations of Kansas Blue Shield.

More babies are being born in hospitals and with a doctor in attendance than ever before, Health Information Foundation reports. In 1935 only 37 per cent were born in hospitals, and 13 per cent of all births were unattended by doctors. In 1956 almost 95 per cent were hospital-born, and doctors attended 97 per cent of all births.

THE MONTH IN WASHINGTON

Editor's Note. The following summary of Washington news was prepared by the Washington office of the A.M.A. for distribution to state and regional medical journals.

By approximately the mid-term point in its first session, the 85th Congress had shown enough interest in health legislation to hold a variety of hearings, but there was no evidence that many major bills would be passed before adjournment.

Actually, it was not until three months after the session opened that the Administration sent up to Congress two bills it regards as important—one would change the doctor draft act and the other would authorize small commercial companies to pool part of their resources to stimulate expansion and experimentation in health insurance.

Even then, the Department of Health, Education, and Welfare had not released its draft of legislation for federal grants to medical, dental, and osteopathic schools for construction and equipment. On this, there was some reluctance to act until Capitol Hill had decided on the administration's bill for U. S. aid to general education.

Of all these bills, indications were that progress was assured on only one, that providing some revised arrangement for the selective draft of physicians, dentists and "allied specialists." The special doctor draft act, in effect for almost seven years, is scheduled to expire on July 1. Because Defense Department insists it still needs special authority to draft physicians and other professional health personnel by professional classification, the alternative was continuation of a modified doctor draft act or changing the regular draft act.

Meanwhile, a number of other bills had been studied at hearings. They include:

Changes in medical aspect of civil aviation regulations. Witnesses are widely divided on this measure that would set up an Office of Civil Aviation Medicine within the Civil Aeronautics Administration and give the Air Surgeon General who would head the office considerably more authority than now is exercised by U. S. medical officials in this field. There was no official sponsorship of this from the federal government level. It was opposed by the Department of Commerce (where CAA is located) and the Civil Aeronautics Board. However, support came from the outside, including testimony from Dr. Jan Tillisch of the Mayo Clinic, Dr. William Ashe, chairman of the department of preventive medicine, Ohio State University, and Dr. Herbert F. Fenwick, president of the Civil Aviation Medical Examiners. Dr. Tillisch headed an AMA *ad hoc* committee that had started a study of the problem, but he testified as an individual.

Veterans medical care. The House Veterans Affairs Committee had held extensive hearings on a bill to further restrict admission of non-service connected cases to Veterans Administration hospitals, but there were no developments beyond that to encourage sponsors of this legislation.

Civil defense reorganization. Here again a wide split developed at the hearings on just how to reorganize the federal government's participation in civil defense. The administration wanted to strengthen the U. S. civil defense arm (the Federal Civil Defense Administration), but without going to the extent of making a cabinet-rank Department of Civil Defense, which is the goal of Chairman Chet Holifield (D., Calif.) of the subcommittee that had studied civil defense for more than a year.

Control of barbiturate and amphetamine drugs. The objective of bills before the House Interstate Health Subcommittee is to extend federal control to take in the manufacture, compounding, processing, distribution and possession of habit-forming barbiturates and amphetamines.

Pressures for economy that had been evident early in the session seemed to lose their effectiveness when Congress really set to work on the budget for the Department of Health, Education, and Welfare. Whereas in first (non-record) votes the House cut scores of items, it simply reversed itself when roll-call votes were demanded in the final go-around.

As an example, no reductions at all were made in funds for the research institutes, $50 million was restored for grants to help build water pollution treatment plants, $1.3 million was restored to the Food and Drug Administration. A $5 million cut in money for general public health grants to states was sustained by the House—but this money will have to be provided later if the House estimate of the extent of the obligation proves too low.

Economy advocates tried without success in the House to cut $21 million off money for the Hill-Burton hospital construction program.

While in theory the Senate is privileged to make its own cuts in a money bill coming to it from the House, in practice the Senators generally restore much of the money cut by the House and occasionally (as last year) vote large boosts over House figures. So the possibility now is for even higher health and medical budgets before the appropriations bills finally are enacted.

Clinicopathological Conference

Case Presentation

The patient for discussion in this conference was a 54-year-old white man who had been in a stuporous state for 12 hours before his admission to this hospital on September 29, 1956, at 8:00 a.m. He died on October 7, 1956, at 12:10 a.m. About a month before his admission he had had fever and chills and he was treated by his physician for a "kidney infection." On September 18 he had had an episode of vomiting and began to lose weight.

In 1946 he had had a laminectomy for a spinal cord tumor at the eighth thoracic segment, which left him with paraplegia. He had been confined to a wheelchair since that time and had had numerous decubiti. He had been incontinent of urine and feces for about seven days before admission. The night before his admission he was found in a stuporous state by his ten-year-old son with whom he lived.

No reliable family history or system review could be obtained.

On admission he was semicomatose, dehydrated, and cachectic. His blood pressure was 80/50; pulse, 100 and rhythmical; respiration, 24 per minute and deep. There were large, maggot-covered decubiti over the buttocks and both greater trochanters, and there was a superficial, necrotic ulcer with extensive surrounding cellulitis on the left thigh. Pitting edema of the left leg was present. The eyes, ears, nose, and throat were considered normal. The neck was supple. Nothing abnormal was found in the examination of the heart and lungs. The liver was palpable three centimeters below the costal margin, and it was hard. There was a first degree rectal prolapse with fecal impaction.

The specific gravity of the admission urine specimen was 1.010. No sugar was found, but there was a heavy cloud of albumin, 5 or 6 white cells, and 10 to 20 red cells per high power field. The red count on admission was 3,180,000 with 9.0 gm. hemoglobin, and the white count was 29,200 with 87 per cent polymorphonuclears and 10 per cent lymphocytes. There was little change in the urine during his hospitalization except for a slight reduction in albumin. The specific gravity ranged from 1.007 to 1.010.

On October 5 the hemoglobin was 6.8 gm.; the white count was 7,900; the platelet count was 132,000, and the reticulocyte count was 3 per cent. A Kahn test was positive with 8 units. Admission electrolytes were: sodium, 140 mEq/L; potassium, 3.8 mEq; chloride, 105 mEq; carbon dioxide, 20.3 mEq. The carbon dioxide was 29 mEq on October 1 and steadily decreased to 13.5 mEq on October 5. Serum electrolytes were determined daily, and the sodium and chloride values remained normal. On October 2 the potassium was 2.7 mEq, rising to 5.6 mEq on October 6. The fasting blood sugar was 128 mg. per cent on admission and 70 mg. per cent on October 4. The creatinine was 2.3-2.0 mg. per cent. The initial blood urea nitrogen was 95 mg. per cent, falling to 57 mg. per cent on October 6. Serum calcium was 4.2, and the phosphorus was 2.2 mEq/L. The serum albumin was 2.8 gm. per cent; globulin, 3.0 gm. per cent; total cholesterol, 88 mgm. per cent with 19 per cent esters; serum iron, 38 mcg. per cent; thymol turbidity, 23 units. The total bilirubin was 0.7 mg. per cent on admission and 1.5 mg. per cent on October 1 with 0.8 mg. per cent direct bilirubin. The acid phosphatase was 0.2 millimol units. The daily urinary sodium excretion rose from 5.9 to 20 mEq, and the potassium excretion increased from 14 to 70 mEq. The histoplasmin complement fixation test was negative. Candida albicans grew on three urine cultures, and pseudomonas, paracolon, and proteus were each recovered once. Two blood cultures were negative.

The patient was given intravenous fluids, and potassium chloride, chloramphenicol, and novobiocin were administered by mouth. He soon began to take fluids orally, and his sensorium cleared, but his temperature rose to 101.5 degrees and remained elevated. His combined oral and intravenous fluid intake was 2,500 to 4,500 ml. daily. His urinary output was 1,800 to 2,200 ml., and his stools were a normal color and contained no blood.

Twelve hours before death there was a sudden drop in the patient's blood pressure to 60/20. In spite of transfusions, digitalization, and general supportive measures, including adrenal steroids, he became worse and died quietly at 12:10 a.m. on October 7, 1956.

Roy R. Hieger (fourth year medical student): Dr. Purinton, was there a prothrombin time determination?

Dr. Lew W. Purinton (resident in medicine): No, there was not.

Mr. Hieger: What was the blood ammonia?

Dr. Purintŏn: It was not run.

Lawrence Hayes (fourth year medical student): Were steroids given during the hospital course?

Dr. Purinton: Only terminally.

Edited by Jesse D. Rising, M.D., and Mahlon Delp, M.D., from recordings of the conference participated in by the departments of medicine, pediatrics, surgery, radiology, and pathology of the University of Kansas Medical Center as well as by the third and fourth year classes of medical students.

Jess W. Koons (fourth year medical student): Did the patient vomit?

Dr. Purinton: Not during his hospital stay.

Richard Helm (fourth year medical student): Was there any evidence of gastrointestinal bleeding terminally?

Dr. Mahlon Delp (moderator): Dr. Weber, do you know of any?

Dr. Robert Weber (internist): No.

Frank J. Kutilek (fourth year medical student): What was the patient's highest blood pressure?

Dr. Purinton: Some blood pressure readings were as high as 100/60.

Lawrence Silvey (fourth year medical student): Do you know anything about his blood pressure before his admission?

Dr. Delp: Do you know anything about this, Dr. Weber?

Dr. Weber: No, I do not.

Mr. Hieger: Was the antibiotic therapy changed during the hospital course?

Dr. Purinton: No. He had the same treatment throughout.

Mr. Hieger: Did he continue to have tachycardia?

Dr. Purinton: Yes, he did.

Mr. Koons: Did he have a convulsion during the hospital course?

Dr. Purinton: I think that he had a grand mal seizure a short time before he died.

Mr. Kutilek: Was a spinal tap done?

Dr. Purinton: No spinal tap was done.

Mr. Helm: What was his temperature curve during the hospital course?

Dr. Purinton: He came in with a normal temperature, and it remained essentially normal for 24 hours. By the end of the second day his temperature was up to 101.6 degrees, and the following day it reached 103.6. On the fourth day it rose to 104 degrees where it stayed until two hours before his death, when it came down to normal.

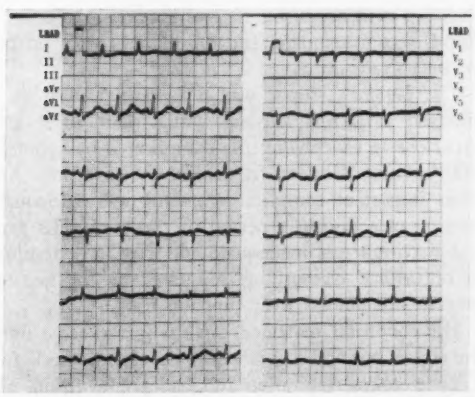

Figure 1. Electrocardiogram made on day of admission.

Mr. Helm: Did you notice any improvement after his admission?

Dr. Purinton: He definitely improved during the first two days.

Mr. Silvey: How was his spleen?

Dr. Purinton: It was not palpable.

Mr. Silvey: Do you think he was an alcoholic?

Dr. Delp: Do you think he was an alcoholic, Dr. Weber?

Dr. Weber: I am not sure. His personal history is poor. He lived alone with his ten-year-old son.

Mr. Hayes: Were his lung fields clear during the hospital stay?

Dr. Weber: During the last 24 hours there were rales.

Mr. Hayes: How much cortisone did he receive?

Dr. Delp: 100 mg. intramuscularly. If there are no other questions, Mr. Hayes will demonstrate the electrocardiograms.

Mr. Hayes: The first tracing was made on the day of admission (Figure 1). The rate is 100; there is normal sinus rhythm, and the intervals are normal. I interpret the arrhythmia in V_1 to be a true extrasystole. The progression of the QRS complexes across the chest leads is normal. The second tracing was made on the fifth hospital day. The rate is 100; there are premature contractions in the V_2 and V_5R. The P-R and Q-T intervals are within normal limits. I interpret this as a normal electrocardiogram with the exception of the premature ventricular contractions.

Dr. Delp: Do you see any change in the T waves of the first tracing?

Mr. Hayes: There is a depression of the T waves.

Dr. Delp: Dr. Crockett, do you have any comments?

Dr. James E. Crockett (cardiologist): There have been some changes in the T vector, with a shift to the left. There are premature ventricular contractions. I do not know what is responsible for them.

Dr. Delp: Do you have any explanation for the T wave changes, Mr. Hayes?

Mr. Hayes: I would consider electrolyte imbalance and cardiac ischemia.

Dr. Delp: Mr. Koons, please demonstrate the x-rays.

Mr. Koons: The first chest film (Figure 2) taken on the day of admission shows the osteoporosis that one might expect. The costophrenic angles are clear; the heart is not enlarged. There is a well defined shadow which I interpret as being a calcific pleuritis. The next chest film was taken on his fourth hospital day. I believe there is no essential change here, but some points need to be clarified: the pa-

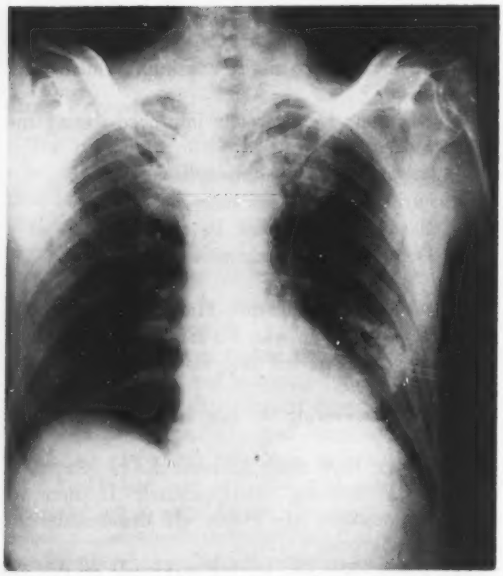

Figure 2. Chest film, taken on day of admission.

tient is rotated, giving the impression of an enlarged heart which I believe is an artifact. I do not see any important changes. The next film shows the marked osteoporosis, and there is evidence of flexion contractures of the thighs. There is evidence of an enlarged liver. I see no kidney or spleen shadows.

Dr. Delp: Dr. Youngstrom, do you have any comments?

Dr. Karl Youngstrom (radiologist): No, sir.

Dr. Delp: Mr. Hieger will now give his differential diagnosis.

Differential Diagnosis

Mr. Hieger: The case we have is that of a 54-year-old white man who was operated upon for a spinal cord tumor ten years ago, and who had been paraplegic since then. One month before his admission he had fever and chills, and he was treated for a kidney infection. On September 19 he began to vomit, and three days later he became incontinent of urine and feces. Twelve hours before admission he became stuporous. On admission he was hypotensive, semicomatose, dehydrated, cachectic, and paraplegic, and he was breathing deeply. Large maggot-covered decubiti were present over the buttocks and both greater trochanters. There was a necrotic ulcer on the left thigh with extensive surrounding cellulitis, and there was marked pitting edema of the left leg. The liver was hard and palpable 3 cm. below the costal margin.

The originating factor in the pathogenic sequence was a spinal cord tumor, which statistically should have been extramedullary, either a meningioma or a neurofibroma. More than 80 per cent of paraplegics die of various complications; chronic renal failure is a common one. In this case certain other causes of coma must be considered. They are trauma, drugs, poisons, infections, brain tumor, and endogenous toxins. Trauma, drugs, and poisons I rule out on the basis of the history. Although this patient had infections in various foci, I think that they were not the cause of his presenting complaint. Brain tumor is unlikely, although there was evidence of increasing intercranial pressure. Endogenous poisoning may cause coma as in diabetes, but the laboratory results do not support this diagnosis, and I rule out cholemia because he was not jaundiced.

The diagnosis of uremia is suggested by the history, supported by clinical impression, and verified by laboratory findings. I shall therefore devote my discussion to complications of paraplegia, the most common and possibly the worst of which are: decubiti, chronic pyelonephritis, renal failure with uremia, poor nutrition, and psychological dependence, all of which this patient had. I believe that he had a cord bladder resulting in residual urine, hydronephrosis, and pyelonephritis, which had been smoldering over a period of ten years with acute exacerbations.

I cannot rule out the possibility of nephrolithiasis, but no stones were seen in the x-rays. I believe that infection precipitated uremia in this patient who already had damaged kidneys. He was in acidosis at the time of death. The acidosis was, I believe, secondary to his excessive vomiting, and resulted in Kussmaul breathing. He was treated with antibiotics and intravenous fluids, the latter of which introduced foreign elements into his blood. The drainage of the bladder by catheter probably contributed to the depression of his blood urea nitrogen. However, he did poorly on this regimen. Fever developed and continued as evidence of his state of toxemia. His urinary output was inadequate, and the kidneys were unable to concentrate urine.

The patient's stupor was, I believe, caused by a number of factors, among which were toxic and hypertensive encephalopathy complicated by hypotension, anemia, dehydration, and severe infection. Another important complicating factor was inadequate nursing care during a period of ten years. His general nutrition was inadequate and may have resulted in nutritional cirrhosis as suggested by the hepatomegaly and altered liver function tests.

His condition remained essentially the same until the eighth hospital day when he developed shock and Cheyne-Stokes respiration and had a convulsion. He died in spite of transfusion, digitalization, and supportive therapy. His death can be explained in a

number of ways. He might have had pulmonary embolization, as he obviously had thrombophlebitis in his left leg. On the other hand he was not cyanotic, had no chest pain, and survived for 12 hours after the onset of hypotension. Hemorrhage with cerebral edema must also be considered, because vascular lesions secondary to uremia and hypertension predispose to this. Cerebral hemorrhage cannot be ruled out, because a spinal tap was not done. The urinary output was good, and I do not think that he had hyperkalemia, nor did he have a myocardial infarction during the final episode. In the absence of edema and tachycardia, I do not believe that he had acute cardiac failure. The terminal episode is best explained by diffuse central nervous system involvement, with focal edema, softening and gliosis, and hemorrhages into the brain substance.

I also believe that he had secondary amyloidosis. The factors complicating this disease contributed to his demise. The hepatic enlargement, abnormal hepatogram and history of long-standing infection with the development of uremia and vomiting are more significant of amyloidosis than of nutritional cirrhosis.

I think this patient had chronic renal disease, uremia, and metabolic acidosis, all secondary to long-standing pyelonephritis which was complicated by secondary amyloidosis. The terminal episode was the result of diffuse encephalopathy and hemorrhage. Lastly, I would like to predict what the pathologists will show us: I believe we will see large, pale, waxy kidneys with evidence of acute and chronic pyelonephritis; a large, waxy liver; passive congestion of the liver, spleen, intestine, and lung; hyperemia and fatty depletion of the adrenals; organic vascular occlusion with focal and diffuse cerebral edema, and actual hemorrhage into the brain.

Clinical Discussion

Dr. Delp: Thank you. Mr. Keith, why did this patient have a convulsion?

Charles R. Keith (fourth year medical student): That can be explained by his prolonged hypertension.

Dr. Delp: Mr. Silvey, what do you think about that?

Mr. Silvey: I agree with that idea.

Dr. Delp: Mr. Koons?

Mr. Koons: I believe that the patient had emboli to the brain.

Dr. Delp: What do you think were the primary foci?

Mr. Koons: I was thinking of the leg. The patient had multiple decubiti, septicemia, and probably impairment of circulation.

Dr. Delp: Mr. Helm?

Mr. Helm: I think that the convulsion can be explained on the basis of general encephalopathy.

Dr. Delp: What do you mean by encephalopathy? That is a vague general term.

John D. Dougherty (third year medical student): I think the patient's hypertension led to cerebral infarct. He had renal hypertension which might lead to cerebrovascular hypertension with infarct and necrosis.

Dr. Delp: Mr. Keith, regardless of what Mr. Dougherty said, you can see in the patient's chart that he definitely had hypotension, not hypertension. How do you account for this hypotension?

Mr. Keith: I can explain it in several ways. He might not have been adequately rehydrated. Also he had toxemia, although his blood was relatively sterile. Finally, secondary amyloidosis may also account for it.

Dr. Delp: Mr. Kutilek?

Mr. Kutilek: I believe this man might have had adrenal insufficiency.

Dr. Delp: How do you make adrenal insufficiency out of this?

Mr. Kutilek: The patient had secondary amyloidosis which involved the adrenals.

Dr. Delp: Let's get back to the appearance of the patient when he came in. He had maggots in his decubiti. Any comment about that?

Mr. Kutilek: I think he had general poor management all the way along, but maggots are good debriders.

Dr. Delp: Now, let us go back to the period ten years ago and do a little guessing about what might have been the trouble with the patient at that time.

Mr. Keith: I believe that he had a benign extramedullary tumor then, which does not have anything to do with the present trouble.

Dr. Delp: Do you think that he lived as long as, or longer than, the average person with paraplegia?

Mr. Keith: After World War I, before antibiotics were developed, none of the large number of paraplegics was alive after five years, but since the advent of antibiotics 85 per cent of a group of paraplegics were alive after five years. This man lived ten years after the development of paraplegia.

Dr. Delp: Of course, there is no evidence that this man had not been receiving some kind of treatment. The amazing thing, however, is that he died. How long did he live after coming to the hospital?

Mr. Keith: He died eight days after admission.

Dr. Delp: Dr. Williamson, do you have any comments to make about this patient?

Dr. William Williamson (neurosurgeon): I did not remember this patient, but one of the relatives recognized me as the surgeon who had operated on him ten years ago. I then remembered that he had

had a meningioma of the spinal cord that was totally removed. It was surprising to me that he became paralyzed after he went home. He should have recovered completely. I could only think that I had either damaged the cord in the removal of the tumor, or the cord was so badly compressed that it could not recover. Because of the desperate condition he was in at the time of consultation, it did not appear safe to do any further neurological examinations such as spinal puncture.

It also occurred to me that this man suffered from complications as a result of lack of follow-up after operation. I had never seen him after he was dismissed from the hospital until I was called in consultation during his final illness. I suppose that it occurred to the family that he would just become paralyzed after he was operated upon; no one thought anything about it. I was the only one who felt disappointed that he was paralyzed as the result of lack of follow-up after an apparently successful removal of a meningioma.

Dr. Delp: Dr. Weber, the students are eager to hear from you.

Dr. Weber: This patient presented a rather difficult problem. He had jack-knife contractions of both legs, and it was impossible for him to lie in bed comfortably. He was admitted in a semicomatose state, and at that time he was afebrile. Part of our management consisted of removing the maggots, and after their removal he became febrile. Cultures of the decubiti revealed that he had staphylococcal infection. Pseudomonas and monilia were cultured from his bladder. We treated him with antibiotics: chloramphenicol, a rather wide spectrum antibiotic, was used, and novobiocin was also administered on the assumption that he probably had hospital organisms.

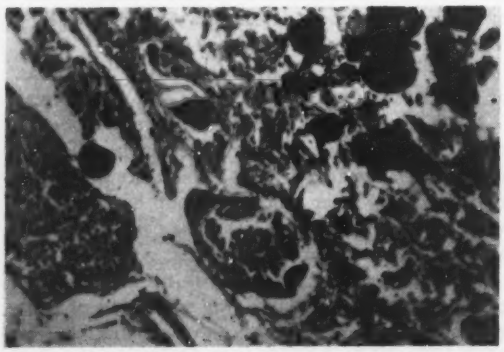

Figure 3. Section of meningioma found at autopsy at level of second thoracic vertebra. Tumor is histologically identical to that removed previously and to the third meningioma at the level of the third thoracic vertebra. Growth consists of islands and strands of meningothelial cells with numerous interspersed psammoma bodies (dark, round calcified bodies). Hematoxylin and eosin stain, X130.

I think he died of hypotension secondary to his toxicity and infection. I expected when he was admitted that we would find renal stones or bladder stones. Here was a patient who had been ill and lying in bed for a period of ten years. I was sure he had severe osteoporosis and possible deposition of calcium in the kidneys and bladder. He also had a cord bladder, hydronephrosis, and renal infection with uremia. I diagnosed this as a combination of renal failure and infection.

The situation was at no time encouraging about rehabilitating the patient because of the serious infections that he already had, and, with the decubiti, it was difficult to handle him. My impression, after seeing the x-rays and noting that he did not have stones, was that he had chronic pyelonephritis and infections secondary to the superficial lesions on the skin. He probably did have bacteremia. Terminally, we used cortisone as a desperate attempt to combat his hypotension.

Dr. Delp: May we hear about the pathology?

Pathological Report

Dr. Harlan Firminger (pathologist): You have already heard the description of the external appearance of this man which was not changed when he came to autopsy.

In the history there was a previous operation nine or ten years ago, at which time a tumor was removed from approximately the level of the eighth thoracic segment. Examination of the specimen revealed a meningioma (Figure 3). The main cellular elements were meningothelial cells growing in the form of whorls, which at times were hyalinized and frequently produced sand-like grains of calcific material.

In the area of the previous operation there was considerable scarring, but there was no adherence of fibrous strands to the spinal cord. If we had not had the history and external scar, we might not have known that anything took place at that level. In the sections of the spinal cord taken below the level of the previous operation, that is at the level of the tenth thoracic segment, stains for myelin sheaths of the nerves revealed perfectly intact posterior columns, but there was loss of the anterior and lateral corticospinal tracts (Figure 4C). At a section higher up, at the level of the first thoracic vertebra, there was degeneration of the fasciculus gracilis. The fasciculus cuneatus was intact (Figure 4A). There was some loss, but not so clearly demarcated, in the spinotectal, spinothalamic and spinocerebellar tracts. The anterior column was intact, and the lateral column was well myelinated. Thus the ascending de-

generation in the region of the first thoracic vertebra and the descending degeneration in the lower thoracic region represented the result of transection in the thoracic region.[2]

It would be logical to conclude that this was the result of the old meningioma; however, at autopsy we found two more meningiomas at the level of the second and third thoracic segments, respectively, which were morphologically identical to the original tumor (Figure 3). The question now arises as to which of the meningiomata really produced the paraplegia. The answer lay in sections taken between the level of the previously excised tumor and that of the meningiomata found at autopsy. A section taken from the level of the fifth thoracic segment (Figure 4B) showed that the fasciculus cuneatus in the dorsal columns were intact, and there was anterior and lateral corticospinal loss, which indicates that the real lesion producing the damage lay above the level of the meningioma that was removed, and that the paraplegia was actually caused by the meningiomas at the second and third thoracic segments.

In addition, there was degeneration of peripheral nerves. Ordinarily we do not expect peripheral nerves to degenerate when there is transection of the spinal cord. A section of the sciatic nerve stained with the myelin stain showed that most of the myelin sheaths were destroyed, indicating peripheral nerve degeneration, too. We attribute this to a nutritive type of peripheral neuritis and not to transneuronal degeneration.

As the result of the lack of stimulation from the nervous system, the muscles of the leg underwent atrophy, and in the section of one of the hamstring muscles one can hardly recognize it as muscle (Figure 4D). There is connective tissue and fat replacing the muscle; here and there are groups of multinucleated muscle cells representing proliferation of sarcolemmal nuclei, but almost no intact muscle.

A section of the base of the decubital ulcer revealed extensive invasion of the tissue by masses of bacteria extending deep into the subcutaneous fat and surrounded by inflammatory cells with a great deal of necrosis.

Mr. Hieger emphasized that urinary tract infection is often the main source of trouble in paraplegics, and the section of his thickened bladder showed areas of ulceration and hyperemia of the mucosa. The necrotic debris in the base of the ulcer contained Candida albicans or something closely resembling it.

The major question that arose in our mind was, "What was going on in the kidneys?" By all odds, and by all that we have learned, it should have been chronic pyelonephritis. However, at the time of autopsy, instead of finding small kidneys, we found that they were large and pale, somewhat resembling amyloid kidneys, as Mr. Hieger suggested. The cortex was markedly thickened and pale. There was some congestion of the cortico-medullary junction with focal areas of congestion within the kidney substance. Microscopically, this was thought to be an interstitial nephritis simply because it did not fit anything else we know. The glomeruli were mostly intact. There were no areas of scarring. Instead there were patchy areas of chronic inflammatory infiltration consisting of lymphocytes, many plasma cells, and mononuclear cells with almost no polymorphonuclear cells participating. There did not seem to be anything acute in most of the lesions, and the striking thing was in the tubules which showed a moderate amount of degenerative change with albuminous casts. This lesion was primarily a nephrosis but with its inflammatory component, for want of

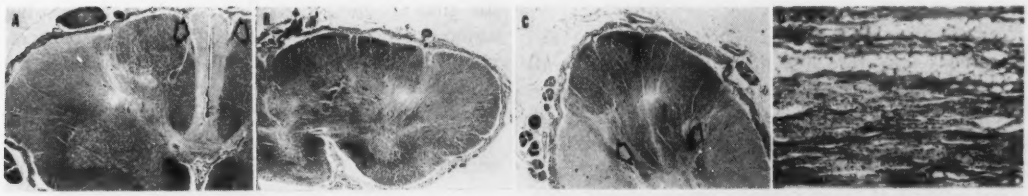

Figure 4A. Spinal cord section at level of first thoracic vertebra. Note loss of myelinated fibers in the fasciculus gracilus of the posterior column between the arrows. The fasciculus cuneatus is intact. Luxol fast blue stain for myelin (appears dark on photograph). X12.

Figure 4B. Spinal cord section at level of fifth thoracic vertebra reveals no loss of myelin in posterior column but some loss in lateral column, as in Figure 4C following and in the anterior column (not shown in 4C). Luxol fast blue stain for myelin. X9

Figure 4C. Spinal cord section at level of tenth thoracic vertebra showing demyelinization of lateral columns indicated by arrows. The posterior column is well myelinated. The anterior column, not shown, also was partially demyelinated. Luxol fast blue stain for myelin. X12.

Figure 4D. Note the atrophy and loss of striated muscle with replacement by connective tissue and adipose tissue. Hematoxylin and eosin. X85.

a better term, we have called it interstitial nephritis. We proposed that this was related in some way to a chronic or subacute lower nephron nephrosis.

There was an ulceration and necrotic material in the cardia of the stomach at its junction with the esophagus. Under high power we found that this contained a mass of fungi resembling Candida albicans with inflammatory reaction about it. The periodic acid-Schiff stain showed the fungus distinctly with underlying reaction to it. It was entirely similar to the lesions in the urinary bladder. This certainly represented a surface type of infestation rather than dissemination by the blood stream.

Finally, the adrenal glands showed marked exhaustion. The adrenal cortex was hardly recognizable. It had a tubule-like formation of cells almost completely devoid of fat and falling apart with loss of cells and edema. We were unable to demonstrate amyloid anywhere.

In summary, this was a man who had a meningioma ten years ago, which, unfortunately, was not the only meningioma he had. He had two others which did not grow notably, but continued to compress the cord and produced his paraplegia. They were identical in morphology with the original lesion, and this raised the question whether or not they could represent dissemination at the time of operation. Kernohan and Sayre[8] reported a case of meningioma in a muscle of the head which apparently was the result of implantation at the time of removal. This is a possibility, but I believe that it is quite unlikely, despite the similarity in morphology. Multiple meningiomata are not at all rare, and I believe that this is what this case represents. It is clearly not a malignant tumor, and it has not disseminated in that manner. There are cases of diffuse meningiomatosis in which one has multiple plaques all through the meninges,[1] but this case certainly does not fit into that category.

As a result of his meningiomata he developed paraplegia, and this was his chief complaint until considerable infection developed. The toxins from his infection must have had a lot to do with the development of his renal disease; perhaps his hypotension also contributed. It was fundamentally a subacute type of lower nephron nephrosis with casts, loss of albumin, loss of tubular function as evidenced by increasing loss of sodium and potassium, which suggests a lack of tubular reabsorption.

Finally, he developed a moniliasis, possibly as a result of the antibiotic therapy. He had a small focus of pneumonia, but I think uremia was the major factor in bringing about his death.

Mr. Hieger: What about the liver?

Dr. Firminger: The liver was large, as you suggested. It weighed over 2,300 gms. It showed very little fat. There was a slight increase in the fibrous tissue in the portal areas, and small numbers of chronic inflammatory cells, chiefly lymphocytes and plasma cells. There was not enough increase in fibrous tissue to call it cirrhosis, and not enough fat to explain the enlargement on the basis of fatty metamorphosis. I do not know exactly why it was enlarged. It was diffusely congested.

Dr. Delp: Dr. Poser, do you have any comments to make about this case?

Dr. Charles M. Poser (neurologist): I think it is worthwhile to emphasize the care of the paraplegic patient, and this applies to any patient with paraplegia. As Mr. Hieger pointed out, since World War II there has been a great improvement in the care of the paraplegic. The general care of the patient is primarily prophylactic; once the decubiti are present they are difficult to manage. Secondly, the metabolic problem is of importance in these patients because the metabolism of protein has to be maintained. They usually have the problem of calcium deposit in sundry places as the result of skeletal decalcification secondary to protein loss in the skeletal stroma. Continuous care, therefore, depends on the control of metabolism, the care of the urinary tract, and physiotherapy. Control of infection is of primary importance, and if this man had been well taken care of there is no reason why he should not be living today.

Dr. Delp: Any further question or comment?

Dr. Williamson: I would be inclined to favor multiple meningiomata instead of transplant because there was no recurrence of the tumor at the site of operation. Furthermore, the lesions were far apart. I believe that he did have multiple meningiomata, and I am sure the upper one was there at the time I operated on the lower one. When we did the myelogram it filled up at the level of the eighth thoracic segment, and we removed the tumor. We did not do another myelogram after surgery, but assumed that it was all right. Had the patient reported after the operation with more complaints, we would certainly have reinvestigated to find out whether there was another tumor higher up, and we would have removed it, because it was surgically curable.

Pathological Anatomical Diagnosis

Two psammomatous meningiomata compressing the spinal cord at the levels of the second and third thoracic segments, with degeneration of the fasciculi gracilis and dorsal spinocerebellar tracts proximal to, and the anterior and lateral corticospinal tracts distal to the meningiomata.

Atrophy and fat replacement of the muscles of the lower extremities, moderate, with flexion contracture of the right hip and knee, advanced.

Osteoporosis of the lumbar spine, moderate.

Emaciation.

Degeneration of the ileoinguinal and sciatic nerves.

Multiple decubiti of the right thigh, the perineum, both buttocks, and over the right greater trochanter.

Acute hemorrhagic and chronic cystitis, moderate, with Candida albicans present in acute ulcers.

Diffuse interstitial nephritis, bilateral, advanced.

Acute passive congestion, all lobes of both lungs.

One focus of acute bronchopneumonia, lower lobe of right lung.

Hypertrophy and dilatation of the heart, 370 gm.

Acute passive congestion of the liver, moderate.

Fatty metamorphosis of the liver, slight.

Lipid depletion of the adrenal cortices, advanced.

Acute ulcerative esophagitis, with fungus organisms compatible with Candida albicans in the ulcerated areas.

Summary

Dr. Delp: This is truly a case to prove the continued value of the autopsy and careful reconstruction of the clinical picture upon a basis of the postmortem findings.

Treacherous multiple foci of meningiomata in the spinal cord assured failure of the original surgery when only one, and the less important lesion at that, was removed. Compounding the difficulty was the failure in follow-up at which time delayed correction might have occurred.

The usual chronic and recurrent urinary tract infections did occur here as is common in patients with paraplegia. However, this can be cited for a minor role only in the final cause of death. It is likely that the patient acquired several hospital organisms which were resistive to antibiotics, and soon had a fulminant bacteremia. The medical shock state following contributed greatly to the tubular damage appearing in the sections of renal tissue. The careful ridding of the decubiti of the maggots may have been the first step in destroying nature's original line of defense.

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American families spent about $950 million in 1956 to pay the doctor, hospital, and other maternity bills for the safer care of mothers and children in more than four million births, according to an estimate by Health Information Foundation. The average cost per family was $225.

COUNTY SOCIETIES

Three guest speakers presented a program on the Jenkins-Keogh bill now in Congress at a meeting of the Sedgwick County Medical Society in Wichita on April 2. Mr. George Powers discussed legal aspects of the bill, Mr. C. Merritt Winsby, C.P.A., spoke on its actuarial features, and Mr. John H. Laffer described its insurance features.

Dr. Carroll P. Hungate discussed "The Role of the Physician in Mass Casualty Care" at a meeting of the Wyandotte County Society in March. For the program on April 16 Mr. Thomas C. Hurst, trust officer of the Commercial National Bank in Kansas City, spoke on estate planning.

A meeting of the Montgomery County Society was held at the Booth Hotel, Independence, in March. Dr. Jack Fair discussed work being done with mentally retarded children at the Parsons State Training School.

Present officers of the Miami County Society are Dr. W. O. Appenfeller, Osawatomie, president, and Dr. George Zubowicz, superintendent of the Osawatomie State Hospital, vice-president.

Dr. Robert W. Weber, of the University of Kansas Medical Center, spoke on "New Antibiotics" at a meeting of the Riley County Society held in Manhattan on March 20.

"Recent Trends in the Law as They Relate to Medicine" was the subject discussed by Mr. Hugh P. Quinn, Wichita, at a meeting of the Shawnee County Society in Topeka on April 1. At the business session the members approved a plan to give polio vaccine to all county residents under the age of 40 as a society project in cooperation with other community agencies.

Members of the Geary County Society were hosts at a meeting of the Golden Belt Medical Society at the Country Club in Junction City on April 11. Dr. G. Bernard Joyce, Topeka, spoke on "Congenital Orthopedic Deformities," and Dr. John F. Christianson, Kansas City; Dr. Robert K. Wallace, Manhattan, and Dr. William E. Mowery, Salina, discussed "Treatment of Thyrotoxicosis."

A meeting of the Central Kansas Medical Society was held at Russell on March 14. Speaker of the evening was Dr. Don R. Miller, of the University of Kansas Medical Center, who discussed "Surgery of the Rectum and Colon."

PHYSICIANS' ACTIVITIES

Dr. Ellis Duncan, assistant professor of surgery at the University of Louisville School of Medicine, has announced plans to open an office for private practice in Burlington.

Three Wichita physicians, **Dr. A. M. Tocker, Dr. Charles DeHaan,** and **Dr. B. E. Stofer,** are authors of a paper, "Primary Pulmonary Leiomyosarcoma," published in the March issue of *Diseases of the Chest.*

Dr. Robert M. Fenton, formerly of Greensburg, is now practicing in Garden City.

Dr. Fowler B. Poling, Wichita, was guest speaker at a recent meeting of the Sedgwick County Medical Assistants' Society. He discussed "The Desirable Personality."

The commissioners of Jefferson County have named **Dr. Robert R. Snook,** McLouth, health officer to replace **Dr. C. J. Bliss,** Perry, who recently resigned.

Dr. William R. Miller, who has been practicing in Axtell, recently moved to Summerfield, a town in which he had been located before going to Axtell.

Announcement has been made by **Dr. Richard E. Hille,** who has been practicing in Garden City since September, that he will soon establish his practice in Lakin. He is a graduate of the University of Kansas School of Medicine.

Dr. Frances Allen, Newton, **Dr. William E. Larsen** of the University of Kansas Medical Center, and **Dr. Robert P. Norris,** Wichita, took part in the program presented at Wichita, May 6 and 7, at the annual convention of the Kansas Society of Medical Technologists.

"Diseases of the Liver" was the topic chosen by **Dr. Samuel Zelman,** Topeka, for an address given to medical technologists of the northeast district of Kansas at a meeting held in Topeka last month.

Dr. A. R. Chambers, Iola, a member of the Kansas House of Representatives, addressed the Kiwanis Club in Iola during the recent legislative session and explained the basic science bill and the healing arts bill then under consideration by the House.

Dr. Mary Glassen, who had practiced in Phillipsburg for 23 years, announced her retirement on March 1. At the same time she announced that **Dr. Thomas F. Taylor,** who had been practicing with her since last summer, is now associated with **Dr. V. W. Steinkruger, Dr. R. J. Koza,** and **Dr. G. L. Osborne** for group practice in Phillipsburg. Dr. Osborne formerly practiced in Clay Center.

A feature story in the *Wichita Beacon* recently described the life of **Dr. L. Clair Hays,** Cedar Vale, who flies a plane to conserve time in his practice, in the conduct of ranching activities, for vacations, and for visiting uranium claims in Wyoming. Dr. Hays flies a Beech Bonanza and has four runways at his private airport on his ranch.

Dr. W. R. Beine, Coffeyville, has been appointed to the Coffeyville Board of Education to fill an unexpired term.

New in Emporia is **Dr. Ronald J. Daniels,** a graduate of Creighton University School of Medicine who has been practicing internal medicine at the Veterans Administration Hospital in Omaha during the past year and a half.

Dr. Arnold H. Greenhouse discussed the cooperation of ministers and doctors in caring for the sick at a joint meeting of physicians and ministers in Garden City recently.

"Mental Health" was the subject discussed by **Dr. Lewis L. Robbins,** of the Menninger Foundation, at a recent meeting of the Woman's Auxiliary to the Shawnee County Medical Society, Topeka.

Dr. Robert E. Stowell, of the University of Kansas Medical Center, recently spent a week at the Armed Forces Institute of Pathology, Washington, as a consultant. He also gave a lecture, "Chemical and Histo-Chemical Changes of Liver Cells during Necrosis."

Plans to practice in St. John after July 1 have been announced by **Dr. Ross Jewell,** a graduate of the University of Kansas School of Medicine who is now serving his internship at Wesley Hospital, Wichita.

Dr. J. R. Bradley recently resumed practice at the Bradley-Waldorf Clinic in Greensburg after completing two years service with the Air Force in Tampa, Florida.

Dr. Thomas G. Orr and **Dr. Stanley R. Friesen,** of the University of Kansas School of Medicine, are authors of a chapter on "Hemorrhagic and Traumatic Shock" in a recently published edition of *Lewis' Practice of Surgery.*

Dr. Paul H. Beckman, a graduate of the University of Tennessee Medical School who has been interning at St. Francis Hospital, Wichita, has announced plans to practice three days a week in Douglass.

A former resident of Grand Island, Nebraska, **Dr. Theodore E. Wade,** recently opened an office in Liberal.

Dr. Homer L. Hiebert, Topeka, was a guest speaker at a refresher course on isotopes given by the Kansas Society of X-ray Technicians at Wichita last month.

Dr. Ralph H. Major, professor emeritus of medicine at the University of Kansas Medical Center, spoke on "How Hippocrates Made His Diagnoses" at the Medical College of Georgia on April 25. The occasion was a meeting of the Georgia chapter of Alpha Omega Alpha.

Dr. Mary Glassen, Phillipsburg, was one of the speakers at a meeting of the National Federation of Press Women at San Antonio on April 18.

A Budapest physician who fled Hungary in February, **Dr. Imre Fenyes,** was brought to Kansas through having heard the name of the late Dr. A. E. Hertzler and is now living in Halstead. He is working in the laboratory at the Halstead Hospital and plans to become licensed to practice medicine here as soon as possible.

A paper, "The Second Stage of Labor," was presented by **Dr. L. A. Calkins,** of the University of Kansas Medical Center, at a meeting of the Tulsa Obstetrical and Gynecological Society on March 13.

Dr. Gerald L. Ward, Ellis, spoke on mental health at a meeting of the Y.W.C.A. at Ellis recently.

Two Lawrence physicians presented a program at a recent meeting of the Cordley Parent-Teacher Association in Lawrence. **Dr. Helen M. Gilles** spoke on nutrition, and **Dr. M. Erik Wright** discussed emotional development.

Dr. Melvin L. Masterson, who has been practicing in Louisburg, has announced plans to move to Paola to practice in association with **Dr. Jack Rowlett** and **Dr. Rex Stanley.**

Dr. Arnold V. Arms, of the University of Kansas Medical Center, has been notified by the ministry of foreign affairs in Santiago, Chile, that he has been appointed honorary consul for Chile in Kansas City.

Plans to move to Lindsborg and to construct an office building there have been announced by **Dr. Deryl D. Fuller,** who has been practicing in Marquette.

A newcomer to Kansas is **Dr. Necati Erdentug,** now practicing in Anthony. Dr. Erdentug, who has studied in England, Norway, Sweden, Denmark, and Turkey, came to Kansas from Nashville, Tennessee.

Dr. John W. Turner, Garden City, spoke on "What's New in Medicine" at a recent meeting of the Cultura Club there.

Dr. J. E. Seitz, WaKeeney physician since 1948, has announced plans to move to Ellsworth to practice in association with three others, **Dr. Richard H. O'Donnell, Dr. Henry S. O'Donnell,** and **Dr. Alfred J. Horejsi.**

A feature story about **Dr. James G. Lee,** Bonner Springs, was published in the *Kansas City Kansan* on March 17 in a column, "Just Lookin' Around." The story told of Dr. Lee's 60 years in practice.

Dr. Chauncey G. Bly, of the University of Kansas Medical Center, discussed cancer research at a recent meeting of the Johnson County unit of the American Cancer Society.

Dr. Bruno Minz, director of research and education at the Osawatomie State Hospital, was recently given the Godard Award by the French Biological Society in Paris. The award, given annually to the

scientist who has contributed most to the fields of biology and experimental medicine, was given Dr. Minz for his work in physiology. He is on leave of absence from the University of Paris, where he is a director of research, and is investigating the possible psychophysiological correlates of the major psychoses. He is devoting part of his time to teaching at the University of Kansas Medical Center, where he was recently appointed professor of pharmacology.

Dr. W. Clarke Wescoe, dean of the University of Kansas School of Medicine, was the subject of a major story in the February issue of *The Palm,* publication of Alpha Tau Omega fraternity. The story was called "Manager of 'Medical City' " and was written by Conwell Carson for first publication in the *Kansas City Star.* Dr. Wescoe is referred to as "ATO's outstanding senior of 1941."

Dr. Edward Crowley, Wichita, presented a paper on maternal deaths in Kansas in 1956 at a section meeting of the American College of Obstetricians and Gynecologists in Dallas last month. **Dr. Jerome Menaker,** Wichita, presided at a luncheon seminar on pelvic congestion syndrome at the same meeting.

Dr. John B. Dixon, who has been practicing ophthalmology with the Wichita Clinic, has announced plans to enter private practice in Mason City, Iowa, on June 1.

Cryptococcosis

(Continued from Page 303)

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Medical Services

(Continued from Page 300)

the physicians practicing therein, are classed as specialists. Wichita has the greatest number and percentage of physicians who are specialists. There is a preponderance of physicians in the surgical specialties, as 425, or 44.8 per cent of the specialists in the state, are in this field, while 389, or 41 per cent, are in the medical field, and 134, or 14.2 per cent, in the other specialties.

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Two-Way Medical Communications

Physicians who attend the A.M.A. meeting in New York in June will be in direct two-way communication with physicians attending the Harvey Tercentenary Congress in London on June 5 through a hook-up arranged by Smith, Kline and French Laboratories, Philadelphia. An underseas cable will carry a discussion, "The Results of Cardiac Surgery."

Participants in New York will include Dr. Michael E. De Bakey, Dr. Alfred Blalock, Dr. John H. Gibbon, Jr., Dr. Frank L. A. Gerbode, and Dr. George E. Burch.

Legislative Key-Man System

As a means of promoting national legislative activities on a state level, the Committee on Legislation of the American Medical Association has selected a key man from each state to serve as a liaison between it and the physicians in his area. Dr. Clyde W. Miller, Wichita, was named to serve for Kansas.

In the future, whenever action is desired, the key man in each state will be contacted directly by a member of the Committee on Legislation. The key man, in turn, will pass on information to members of a legislative committee in his state.

DEATH NOTICES

Lindley Edgar Strode, M.D.

Dr. L. E. Strode, 84, who had practiced in Girard more than 56 years, died at his home there on February 27. A graduate of Hahnemann Medical School in Chicago in 1899, he opened his first office in Brazilton and practiced there for a year before moving to Girard. He was a member of the Crawford County Society.

Claude E. Burtch, M.D.

An honorary member of the Osborne County Society, Dr. C. E. Burtch, 82, died at his home in Portis on March 7 after having been ill for a year. He was graduated from University Medical College, Kansas City, in 1904 and began practice immediately at Athol. Five years later he moved to Paradise, and two years after that transferred his practice to Portis, where he built a hospital in 1925. He was especially interested in literature and music, and for a time he conducted an orchestra and taught instrumental music.

Karl C. Haas, M.D.

Dr. K. C. Haas, 70, a Kansas City physician who also served as vice-president of the Kansas City Board of Education, died on March 13 at the University of Kansas Medical Center, where he had been a patient for two weeks. A graduate of Eclectic Medical University, Kansas City, Missouri, in 1910, Dr. Haas took postgraduate work at the Kansas City College of Medicine and Surgery. He practiced in Hays and Victoria before opening his office in Kansas City in 1917. He was a member of the Wyandotte County Medical Society and of the American Academy of General Practice.

Schools, offices of the education system in Kansas City, and libraries there closed as a tribute to Dr. Haas during the time of his funeral services. He had been a member of the Board of Education since 1940 and had been vice-president since 1947.

Friedrich W. Tretbar, M.D.

Death came to Dr. F. W. Tretbar, 76, an honorary member of the Stafford County Medical Society, on March 18 after an illness of two years during which he was hospitalized at the Stafford Hospital. He had practiced in Stafford since 1910, and for five years preceding that date, after his graduation from Kansas City Medical College, had practiced in Hudson. He served nine years on the Stafford Board of Education, was a charter member and first president of the Lions Club in Stafford, and was active in many church and civic affairs.

Mark L. Bishoff, M.D.

Dr. M. L. Bishoff, retired chief surgeon of the hospitals of the Santa Fe system, died on March 19 at Topeka, where he had made his home. He was an honorary member of the Shawnee County Society. He was graduated from University Medical College in Kansas City in 1902 and immediately began work with the Santa Fe Hospital Association, serving as assistant surgeon at the Topeka hospital. From 1913 to 1919 he was in charge of the railroad hospital at Fort Madison, and he returned to the Topeka institution in 1919 to serve as chief administrator. He was chief surgeon for the road from 1938 until his retirement in 1947. Dr. Bishoff was a fellow of the American College of Surgeons.

Robert Lee Graham, M.D.

A doctor who had specialized in pathology at two Topeka institutions, Dr. R. L. Graham, 48, was found dead at his home in Topeka on March 22. He practiced first in Topeka at Topeka State Hospital, later serving as pathologist at Winter Veterans Administration Hospital. He was a graduate of Johns Hopkins University School of Medicine, class of 1937, and had been located at Beverly Hills before moving to Kansas.

Eugene E. Hawk, M.D.

Dr. E. E. Hawk, 35, Wichita physician and member of the Sedgwick County Society, was killed in an airplane crash in Harper County on March 23. A graduate of the University of Kansas School of Medicine in 1953, Dr. Hawk served his internship at St. Francis Hospital in Wichita and then began practice there in association with Dr. A. F. Wittman. He was in military service during World War II.

Pulmonary Edema

A Study of Its Pathogenesis and Review of the Literature

DARRELL D. FANESTIL, ***Kansas City***

The purpose of this paper is to review the recent literature on the pathogenesis of pulmonary edema. Each of several factors important in pulmonary edema will be discussed individually; then a correlation of these factors, such as is possible at this time, will be attempted.

The problem of pulmonary edema is unusually complex because of the large number of variables which can produce it experimentally and because of the variety of clinical conditions which apparently may cause it. Because of this complexity, there was disagreement among early students of the problem. Much of this is now being resolved because of the recognition of the distinction between the primary or immediate, and the secondary or remote or indirect causes.[53] For example, at one time emphasis was placed upon "neurogenic" factors with little regard to the mechanisms by which the nervous factors themselves might operate.

Pulmonary edema has been produced by a variety of procedures in the laboratory. Intra-tracheal injection of milk or milk substitute,[40] inspiratory or expiratory resistance,[22] intra-cisternal injection of "fibrin" (thrombin and fibrinogen mixture),[8] intravenous infusions under eupoxic and hypoxic conditions,[15] adrenaline,[28, 57] intra-peritoneal injection of thiosemicarbazide,[52] ammonium salts,[36] craniad injection of fluids under high pressure into the common carotid arteries,[34] the production of hemorrhagic shock,[18] alkalosis induced by intravenous sodium carbonate and sodium bicarbonate mixture,[30] bilateral cervical vagotomy,[19] injection of irritating substances into either ventricle of the heart,[12] electrolytic lesions of the rostral hypothalamus of rats,[20] and inhalation of noxious gases,[7] are all procedures that have been used successfully in the experimental production of pulmonary edema.

Luisada[32] has listed the wide variety of clinical conditions in which pulmonary edema occurs:

A. Heart disease. This includes rheumatic or luetic involvement of the valves, arterial hypertension, mitral stenosis, coronary thrombosis, and diffuse sclerosis of the myocardium. "Instances of these diseases comprise the greatest number of cases of acute pulmonary edema observed clinically in adults."[32]

B. Diseases of the central nervous system. This includes trauma to the skull, diseases of the brain, and stellate ganglion manipulation.

C. Diseases of the lungs. Inhalation of toxic gases, pneumonia, pulmonary infarction, inspiratory obstruction, drowning, and rarely thoracentesis have been accompanied by pulmonary edema.

D. Others.

1. Allergy. "The possibility of angio-neurotic edema of the lungs cannot be ruled out."[32]
2. Stimulation of hollow viscera, especially the stomach. This was noticed mainly by early observers, and the possibility that other diseases might have caused the edema must be considered.
3. Uremia.
4. Thyroid crisis.
5. Beri-beri.
6. Pregnancy.
7. Anesthesia with ether containing peroxide contaminants.[50]
8. In conjunction with insulin shock therapy.[13]
9. Pressure on an enlarged liver in a failing heart case.[37]
10. In salicylate therapy of rheumatic heart disease.[51]
11. Intravenous infusions of saline, especially in patients with cardiac, pulmonary, or renal disease.[1]

History

According to Luisada, pulmonary edema was probably first described by Maloët in 1752. A complete description of the clinical picture was given by Devay, Rilliez, and Barthez from 1850 to 1860. In these same ten years, Virchow and Cohnheim and Lichteim started research on lung edema.[32] In 1878 Welch caused lung edema of the rabbit either by ligature of the aorta or by compression of the left ventricle. This work resulted in the "back-pressure" or "left-ventricular failure" theory: "A disproportion between the working power of the left ventricle and of the right ventricle of such character that the resistance being

This is one of a group of theses written by students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be the best by the faculty at the school. Mr. Fanestil is now a third year student at the school.

CONFIRMED THERAPEUTIC UTILITY

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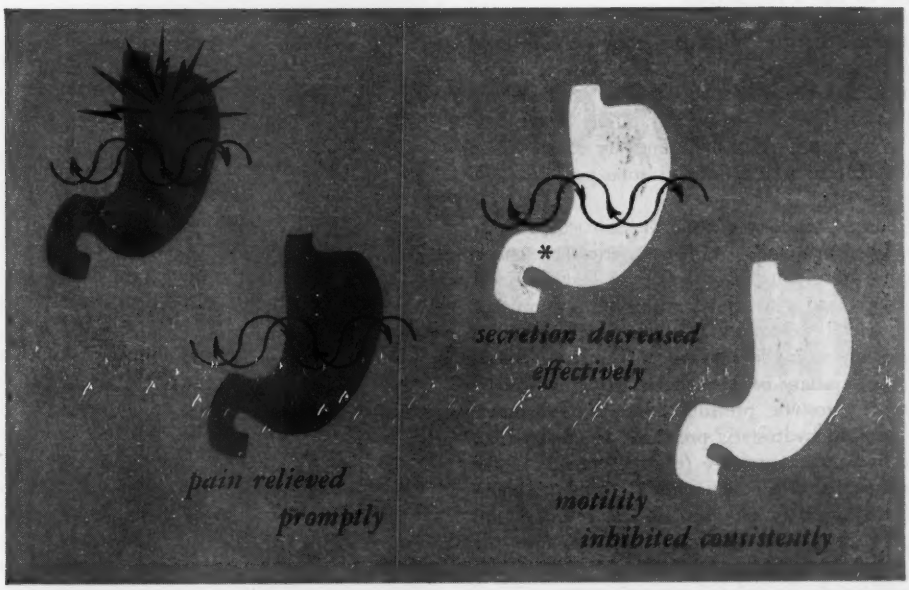
A Primary Drug in Peptic Ulcer



Among the many clinical indications for Pro-Banthine (brand of propantheline bromide), peptic ulcer is foremost. During treatment, Pro-Banthine has been shown repeatedly to be a singularly valuable agent when used in conjunction with diet, antacids, sedation and psychotherapy as required. Lichstein and his associates* report that Pro-Banthine "proved almost invariably effective in the relief of ulcer pain, in depressing gastric secretory volume and in inhibiting gastrointestinal motility. The incidence of side effects was minimal. . . ."

The therapeutic utility and effectiveness of Pro-Banthine in the treatment of peptic ulcer are repeatedly confirmed in the medical literature. Dosage: One tablet with each meal and two tablets at bedtime. G. D. Searle & Co., Chicago 80, Illinois, Research in the Service of Medicine.

*Lichstein, J.; Morehouse, M. G., and Osmon, K. L.: Pro-Banthine in the Treatment of Peptic Ulcer. A Clinical Evaluation with Gastric Secretory, Motility and Gastroscopic Studies. Report of 60 cases, Am. J. M. Sc. *232*:156 (Aug.) 1956.

SEARLE

the same the left heart is unable to expel in a unit time the same quantity of blood as the right heart."[55] This results in pulmonary congestion and exudation of fluid from the capillaries.

As experimental and clinical evidence accumulated, this theory gradually became less accepted.[32] In its place came the neurogenic theory which postulated that the edema was not mechanical but neurogenic in production. Steeves[48] has pointed out that while many clinicians adhere in thought to the mechanical theory, they apply the teachings of the neurogenic theory by using morphine as a therapeutic agent of choice.

Factors in the Production

In general, edema may arise from (A) reduced oncotic pressure of the plasma, (B) obstruction of the lymph channels, (C) an increase in capillary permeability, or (D) an increase in capillary blood pressure.[6] In the specific case of pulmonary edema, these general factors as well as intra-alveolar pressure must be considered.

A. Reduced capillary oncotic pressure could cause pulmonary edema. Under normal conditions the oncotic pressure of plasma is greater than that of tissue fluid. This, then, is a factor favoring fluid movement into the blood stream. Near the arterial end of a capillary, fluid with a low protein concentration moves out of the capillary because the effective hydrostatic pressure (hydrostatic pressure of the blood minus the tissue fluid hydrostatic pressure) is greater than the effective oncotic pressure (oncotic pressure of the blood minus that of the tissue fluid). Near the venous end of the capillary this situation is reversed and fluid poor in protein (the protein goes into the lymph) comes back into the blood.[6] Any factor which reduces the oncotic pressure of plasma will reduce the effective oncotic pressure and, therefore, more fluid will move into and less fluid will be taken up from tissue—in other words, edema may result.[6]

Since it is not the purpose of this paper to discuss factors which can decrease the oncotic pressure of plasma, they will be mentioned only briefly. Because the major portion of the oncotic pressure is due to albumin,[10] hypoalbuminemia is the greatest factor in producing a low oncotic pressure. The major causes of hypoalbuminemia are:

1. Excessive loss of albumin, as in urine, edema fluid, or inflammatory exudates.
2. Inadequate protein supply due to dietary protein insufficiency, vomiting, or diarrhea.
3. Impaired synthesis, as in liver dysfunction, chronic infection, severe anemia, or cachexia.
4. Sudden plasma dilution, as with massive intravenous saline infusions.[10]

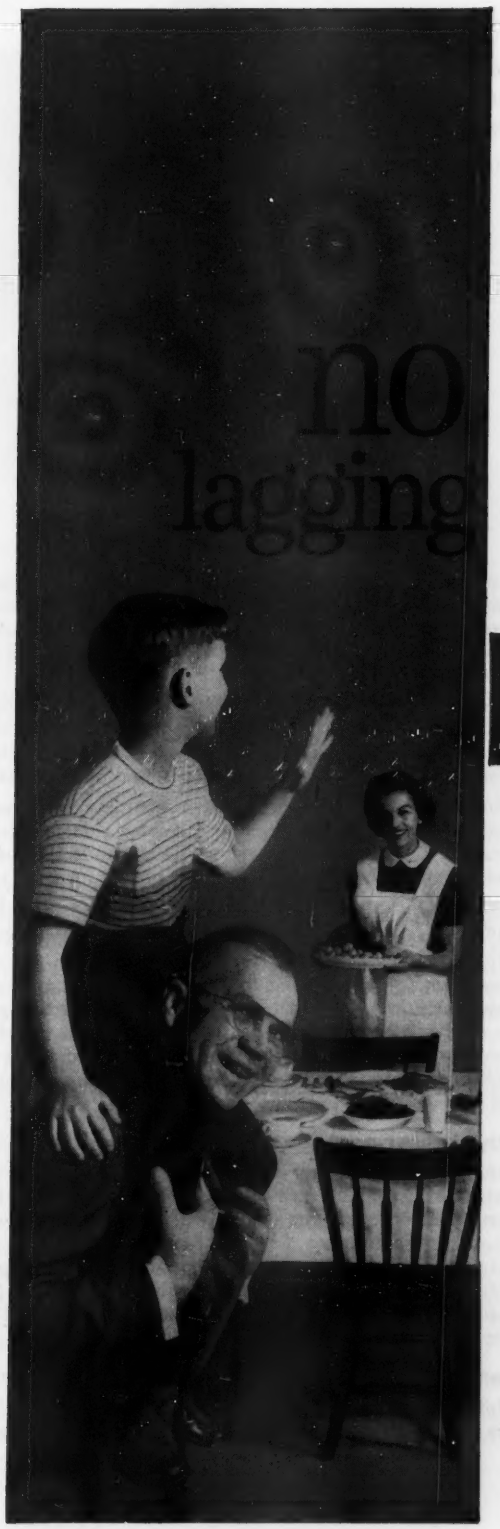
Moran[40] has shown that in the lungs another factor may be important in the reduction of effective oncotic pressure. This investigator showed that intratracheal injections of carbohydrate solutions (12½ grams per cent or stronger) would cause pulmonary edema in rabbits and guinea pigs. As he pointed out, the production of edema apparently results because the oncotic pressure in the alveoli due to the carbohydrate solution is sufficient to overcome that of the plasma in the capillaries. It can also be concluded from this work that any fluid in the alveoli with oncotic pressure may enhance edema formation—indeed, edema itself would enhance more edema.

B. Greater negative intra-alveolar pressure may be a factor in pulmonary edema production. Contrary to other tissues, an important factor opposing the high oncotic pressure and low hydrostatic pressure in the lung capillaries is the negative pressure in the thorax during inspiration.[17] Taylor[6] gives the changes in pressure within the lung: during ordinary quiet respiration, pressure on inspiration is about minus two millimeters of mercury and on expiration it is about plus three or four millimeters; however, maximal negative pressure developed by forced inspiration is from minus 40 to 50 millimeters, and maximum expiratory effort against a closed glottis is from plus 10 to 40 millimeters.

Drinker[17] used a normal animal not subjected to circulatory strain (such as exercise or cardiac incompetency) but subjected to a slight degree of anoxia and resistance to inspiration in an attempt to determine if greater negative inspiratory pressure alone could cause pulmonary edema. He found that the animal could avoid serious pulmonary edema for a long time, but that there were alterations in the lung suggestive of pulmonary edema. Certainly, in the dyspneic individual, the greater negative pressure is a factor to be considered.

C. Decreased lymph drainage or poor ability to handle increased lymph formation may enhance edema formation. "Obstruction to the outflow of lymph from the tissue spaces may cause pronounced edema. . . ."[6]

Drinker says that lymph capillaries in the lung do not surround the alveoli, but end just short of the atria. Thus, alveolar septa must become engorged with fluid before lymph can flow. Because of this, excess fluid filtered out of capillaries can enter the alveoiar air spaces about as easily as it can spread inside alveolar tissue to the lymph capillaries. Drinker believes that normally some fluid does enter the alveoli. This fluid is either coughed up, absorbed directly into the lymphatics, or the protein hydrolyzed and then absorbed into the lymphatics. While blockage of lymphatics may cause edema, even a decrease in the



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This is not to suggest, of course, that side effects will not occur with Harmonyl—as with any potent therapeutic agent. But the mildness of side effects, in the few instances in which they have been reported, suggests Harmonyl as a drug of choice in conditions ranging from mild anxiety to major mental illness and in essential hypertension.

Why fewer and less severe side effects?

Some investigators suggest that the evidence of less parasympathetic effect with Harmonyl in animals might also be true in man. In chronic toxicity studies with Harmonyl this was manifested by less diarrhea, "bloody tears" and ptosis in rats than was observed with the same dosage of reserpine. Dogs also exhibited milder side effects—in particular, diarrhea. No organ toxicity or hematological change was observed with Harmonyl over a wide dosage range.

Harmonyl as a tranquilizer

While Harmonyl's safety is most impressive, clinical investigators reported other notable characteristics for this wide-range

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tranquilizer. For instance, following an eight-month study of chronic, hospitalized mental patients, Ferguson[1] reported:

- Harmonyl benefited at least 15% more overactive patients than oral reserpine.
- Harmonyl was more potent in controlling aggression, requiring only one-half to two-thirds the dosage of reserpine.
- A number of patients experiencing side reactions on reserpine were completely relieved when changed to Harmonyl.

In his summary Ferguson concluded: "*The most notable impressions were the absence of side effects and relatively rapid onset of action with Harmonyl.*"

Harmonyl in hypertension

Hypertension studies show that the average reduction in blood pressure obtained with Harmonyl compares closely to that obtained with reserpine. The tranquilizing effect of the two drugs also appeared similar, except that few cases of giddiness, vertigo, sense of detached existence or disturbed sleep were observed with patients receiving Harmonyl.

Dosages In mild anxiety, as little as 0.1 mg. of Harmonyl a day may be effective. In institutionalized psychiatric patients, not less than 2 to 3 mg. a day is likely to be beneficial.

In mild essential hypertension, treatment may be started with one 0.25-mg. tablet three or four times a day. After about ten days (or sooner, depending upon response), dosage may be reduced. A maintenance dose of 0.25 mg. daily is often sufficient.

Precautions, As with other forms of rauwolfia, Harmonyl must be used cautiously in peptic ulcer and epilepsy and in patients about to undergo surgery or electroshock treatment. Despite infrequent reports involving depression, patients with a history of depressive episodes should be watched carefully.

Professional literature is available upon request.

Supplied: Harmonyl is supplied in 0.1-mg., 0.25-mg. and 1-mg. tablets.

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Reference: 1: Ferguson, J. T.: Comparison of Reserpine and Harmonyl in Psychiatric Patients: A Preliminary Report, Journal Lancet, 76:389, December, 1956.

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amount of lymph flow from the lungs without a decrease in lymph formation may cause more than normal amounts of fluid to be found in the alveoli.

Beattie[5] has pointed out a factor in decreasing lymph flow. Since lymphatic vessels from the lung empty directly into systemic veins, an elevated venous pressure will have an adverse effect on the reabsorption of fluid into lymphatics and, consequently, more alveolar fluid would form. This fluid, then, in addition to being the start of edema, could enhance further edema production by decreasing the effective oncotic pressure and by increasing blood capillary permeability due to anoxia.

In addition to simple obstruction of lymph flow, pulmonary edema may be enhanced by increased lymph formation. Drinker[17] has shown that the lymph drainage from the lungs of the dog (except for a small area near the left apex) is to the systemic circulation via the right lymphatic duct. He believes that practically the same situation exists in humans. This, according to Drinker, is important, for the right lymphatic duct is short, has a sharp curve, and is not too large—in short, it is a "bottleneck." Therefore, an increased lymph flow from the lungs may not be able to enter the systemic circulation as fast as it is produced. This results in the same thing as lymphatic obstruction—an increased tendency for pulmonary edema formation.

However, there is not agreement upon the importance of lymph flow in the genesis of lung edema. Visscher,[53] for example, believes that the increased lymph flow found in association with pulmonary edema is a consequence rather than a cause of the edema.

D. An increase in lung blood capillary permeability would cause pulmonary edema. Anoxia, neurogenic factors, and toxins have been suggested as being responsible for such an increase in permeability.

Drinker has shown that the amount of lymph flowing from the right lymphatic duct of dogs increases when the animal is hypoxic. This he supposed was an important factor in the production of pulmonary edema and further, that the cause of an increased lymph flow was an increase in the permeability of the lung blood capillaries. In general, hypoxia probably causes an increase in capillary permeability. However, Drinker claims that capillaries in some parts of the body are rendered more permeable than others by the same hypoxic state and that the lung capillaries are "significantly assailable" by hypoxia. Further, he believes that the lung capillaries in the alveoli also vary from other capillaries in that they are dependent upon the alveolar air for their oxygen supply rather than upon arterial blood. Because of these two special features of pulmonary capillaries and other evidence in his experience, Drinker said, "It is my belief—I cannot say conviction—that . . . (pulmonary edema) . . . depends more upon alterations in the permeability of the lung capillaries than upon complicated pressure relations in the pulmonary circulation."[17]

Many factors may cause alveolar anoxia: (a) a portion of alveoli are normally collapsed, (b) small mucous plugs may prevent oxygenation, (c) distended pulmonary capillaries can reduce the volume of alveolar air spaces, and (d) excess filtrate or aspirated fluid can occupy alveolar spaces.[41]

Pullen,[41] however, has pointed out that anoxia does not necessarily produce edema. Wiggers concurs: ". . . very low oxygen tensions have been tested innumerable times both on animals and man and do not seem to produce pulmonary edema."[56]

Nevertheless, it is important to mention that Drinker believes that "anoxia begets anoxia" and, consequently, edema. As soon as alveolar capillaries begin to leak excessive fluid, from whatever cause, the barrier between their walls and alveolar air becomes thicker; therefore, the capillary is even more hypoxic. This causes further transudation of fluid—and on around the cycle. "The presence of free alveolar fluid and foam (which is characteristic of pulmonary edema) increases the diffusion distances for gas exchange and also greatly impedes alveolar ventilation. Both of these effects are serious, but the latter is calamitous and is responsible for the rapidly fatal characteristic of acute lung edema."[53]

We may conclude, therefore, that anoxia probably does increase capillary permeability. Whether anoxia by itself can cause pulmonary edema is disputed, but experimentally it does not.[15, 56] However, it may enhance further edema formation once the process has already started.

A neurogenic cause for an increase in pulmonary capillary permeability has also been postulated. Cameron and De[8] state that respiratory excursion, carotid blood pressure, and right atrial pressure undergo similar changes after severe tracheal obstruction, pulmonary embolism with starch granules, and intracisternal injection of "fibrin" with or without vagotomy, but that only the fibrin without vatogomy is followed by pulmonary edema. They then suggest that neither asphyxia nor mechanical disturbances in pulmonary circulation plays an important part in the genesis of lung edema, but that the permeability of lung capillaries may be modified by the outflow of stimuli from the brain via the vagi so that transudation of fluid from the pulmonary vessels is sufficient to produce edema.

Another group of agents, toxic substances, has been suggested as being important in increasing permeability. Examples of "toxic substances" are lethal gases[56]

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and toxic agents carried by the blood, such as bacterial split-products or histamine-like substances from tissue destruction.[16]

Thus, in 1949, Wiggers concluded that ". . . . increase in permeability of pulmonary capillaries seems to be the primary factor in the production of pulmonary edema, but the processes by which such damage comes about are obscure. Anoxia, chemical toxins, and neurogenic factors have been suggested but not established."[56]

E. An increase in the pulmonary capillary hydrostatic pressure may be a factor in the genesis of pulmonary edema. Several mechanisms have been suggested to account for such an increase in pressure: (a) pulmonary vein or venule constriction, (b) increased pulmonary blood volume, (c) left ventricular failure, (d) pulmonary arteriolar dilatation, and (e) the effect of the bronchiolar musculature on the pulmonary circulation.

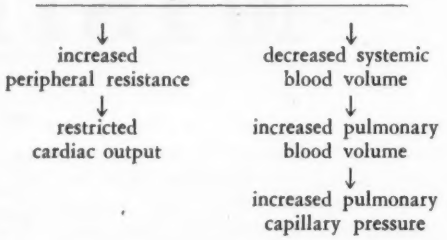
a. Pulmonary vein or venule constriction could conceivably cause an increase in capillary pressure. However, it is doubtful if much constriction ever occurs in the pulmonary veins.

b. Increased pulmonary blood volume as a cause of increased capillary pressure.

Courtice and Korner[15] did experiments in 1952 designed to establish the validity of the experimental observation of and the conclusions drawn from those observations by Drinker, referred to previously. They showed that prolonged and massive intravenous infusions are necessary to produce pulmonary edema in the eupoxic animal, but that hypoxia significantly decreases the amount and length of infusion necessary. Their results showed that hypoxia causes an increase in peripheral resistance and an increase in systemic venous pressure. They then suggested that the factors causing the increased peripheral resistance also cause the redistribution of the blood so as to decrease the peripheral blood volume, increase the volume of the venous blood (and, therefore, the rise in venous pressure), and increase the volume of blood in the expansile pulmonary vascular bed. Thus, although hypoxia will not produce pulmonary edema by itself, it will cause a redistribution of blood so that a small infusion may distend the pulmonary capillary bed enough to cause the capillary pressure to rise over the oncotic pressure. From their extensive experiments they concluded that hypoxia does not cause a selective increase in permeability of lung capillaries, that their (as well as Drinker's) experimental results can be explained on the basis that pulmonary edema is produced when the pulmonary capillary pressure rises enough to overcome the oncotic pressure of the blood, and that such a pressure relation develops more quickly in hypoxia.

These investigators were influenced in their conclusion by the work of the Sarnoffs, who studied hemodynamics associated with intra-cisternal injection of "fibrin." Using this technique, they were able to produce pulmonary edema, as had Cameron and De. The Sarnoffs, however: (a) were unable to prevent the development of the edema by vagotomy,[46] (b) noted that the action of intra-cisternal fibrin was not due to an increase in pressure of the cerebrospinal fluid, but probably was due to a non-specific irritation of medullary centers, such as the cardio-vascular center,[46] (c) noted that the injection is followed by (1) a mean increase of 95 per cent in the peripheral vascular resistance,[47] (2) systemic hypertension and decreased cardiac output,[47] (3) an approximate doubling in the volume of blood in the pulmonary circulation,[47] (4) a gross increase in the size of the left auricle and engorgement of the pulmonary veins,[46] and (5) an increase in pulmonary vascular (artery, vein, and left atrial) pressure with no increase in resistance (and, therefore, an inference of no change in pulmonary vasotension).[49] They held that the increase in peripheral resistance was due to peripheral vasoconstriction. They outline the

effects of this systemic vasoconstriction:[45]



The systemic vasoconstriction, they believe, is mediated by nervous control, for ganglion blocking agents prevent the vasoconstriction and the edema.[44]

Because of their findings, the Sarnoffs entered a new term in the literature, *neurohemodynamic pulmonary edema,* which they define as ". . . that state wherein an increase in the rate of the transfer of fluid from pulmonary capillary to the extra-vascular space of the lung is brought about by an increase in pulmonary capillary pressure, which in turn is brought about either directly or indirectly by nerve impulses."[44]

Tennekoon produced pulmonary edema in rats by intraperitoneal injection of thiosemicarbazide. Because this procedure causes an increase in systemic blood pressure followed by lung edema (both were preventable by sympatholytic drugs) and because thiosemicarbazide fails to produce edema in a heart lung preparation, he believes that this edema is a result of systemic vasoconstriction causing a "short-circuiting"

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of the blood to the pulmonary circulation and that this is mediated through the sympathetic system.[52]

It has been known since 1904[39] that adrenaline could cause pulmonary edema. At first this was explained by Welch's hypothesis.[4] In 1932, however, Hochrein and Keller demonstrated that epinephrine injection was followed by an apparent increase in pulmonary blood volume during the systemic hypertension. Hamilton et al.[23] in 1938 attributed the edema to back pressure from the left ventricle. However, they did describe an "intense systemic vasoconstriction" following the epinephrine injection. In the same year, Barach et al. said, "Our evidence suggests, to our mind, that the high capillary blood pressure is the significant factor in the pathogenesis of pulmonary edema due to adrenalin."[4] Stone and Lowe[49] agree with the last two sources mentioned—that high pulmonary circulatory pressure can account for edema production—but offer no mechanism for the production of this pressure increase, except by noting that it and the edema are prevented by adrenergic blocking drugs.

In 1953 Korner[31] produced pulmonary edema by infusion of nor-adrenaline. He found a rise in systemic venous pressure and an increase in pulmonary blood volume and attributed these findings to a vasomotor redistribution of blood.

A clinical condition in which an increase in pulmonary capillary pressure occurs is mitral stenosis where the outflow of blood from the lungs is impeded. Sympathetic stimulation, caused by excitement, exertion, cold, anger, or fright is followed by (a) tachycardia, which shortens diastole and impairs emptying of the left atrium, and (b) vasoconstriction and resultant redistribution of blood toward the lungs.[35] Both of these increase the pulmonary vascular pressure.

c. The left ventricular failure hypothesis is not widely accepted by investigators in the subject at this time. It may have a role in the development of some pulmonary edemas, as mentioned above in connection with mitral stenosis. However, it generally does not agree with the experimental findings. For example, the Sarnoffs (a) were able to produce an elevation of the left atrial pressure even while systemic blood flow increased, and (b) showed that dilatation of the systemic vascular bed prevented edema formation even though systemic resistance was maintained at a high level by aortic compression.[45]

d. Pulmonary arteriolar dilatation. There has been much debate and disagreement on the role of vasomotor fibers to the pulmonary circulation. Wiggers, in 1949, concluded that "a critical review of all the experimental evidence leaves no doubt as to the existence of vasomotor nerves to the pulmonary vessels in mammals and further strongly indicates that vasoconstrictor impulses travel chiefly over the sympathetic system."[56] However, this same author continues, "Most physiologists agree that the vasomotor apparatus is not important in controlling blood flow or pulmonary vascular resistance."[56] Nevertheless, Aviado et al.[3] demonstrated a dilatation of pulmonary arterioles by hypoxia. Luisada[35] evidently interpreted this as being due to stimulation of the carotid bodies by the hypoxia and a reflex dilatation of the arterioles. It is true that Luisada and Sarnoff[33] in 1946 reported prevention of pulmonary edema by interruption of the reflex path from the carotid sinuses. However, this is hard to reconcile with the report of Wassermann and Goodman[54] in the same year that prevention of pulmonary edema had been accomplished by digital pressure in the carotid body area. If arteriolar dilatation does occur, this would increase the pressure in the pulmonary capillaries.

e. Rodbard[42] postulated another factor that influences the pulmonary circulation, the bronchiolar musculature. During inspiration the bronchioles dilate actively and intra-alveolar pressure falls. An index of this is seen in the pulmonary diastolic pressure. Adrenergic influences also dilate the bronchioles, reducing the intra-alveolar and pulmonary diastolic pressures. "If this effect is marked and prolonged, pulmonary congestion and edema may ensue as a result of the inhibition of the protective bronchiolar muscular tone."[42]

The discussion of one final experimental method of producing pulmonary edema will be helpful. The ammonium ion causes pulmonary edema. This procedure resembles epinephrine-produced edema in many respects.[36] The Koenigs say that one apparent point of difference is that central nervous system depressants (such as morphine or ether) inhibit epinephrine-produced edema, but not ammonium-produced edema. This finding, however, is not substantiated by MacKay, Jordan, and MacKay[36] or by Cameron and Sheikh.[9] There is also disagreement about the mechanism involved.

Cameron and Sheikh obtained no evidence of serious hemodynamic disturbances and "conclude that lesser degrees of pulmonary edema develop in the rabbit after the intravenous injection of ammonium chloride without hemodynamic disturbance."[9] They suggest that capillary permeability for protein is increased by the ammonium ion for they say the edema fluid has a high protein content from the beginning. Tennekoon[52] found that ammonium chloride produces pulmonary edema in the heart-lung preparation and agrees with Cameron and Sheikh that there may be a direct action on the lung capillaries by the ammonium ion. Sarnoff and Kaufman,[43] however, found

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that ammonium chloride intra-peritoneally injected in cats caused left auricular and vena caval pressure to rise significantly and that systemic arterial pressure was normal or lowered. They feel that their work does not preclude the possibility of other mechanisms operating, but that it does suggest that failure of the left ventricle may play a role in the development of the edema.

Jaques introduced another aspect of the problem. He reports that when pulmonary edema is induced by ammonium ion in rats, a depletion of pulmonary histamine occurs.[27] The released histamine does not enter the edema fluid in appreciable amounts but seems to be carried in the blood. The same investigator found that the histamine content of rat lungs increases with the age of the rat and, further, that the incidence of pulmonary edema produced by ammonium ion is strongly correlated with lung histamine levels.[28] He believes that the ammonium ion causes the adrenals to produce adrenaline, which in turn releases the histamine from the lungs, and edema follows. I can only suggest that this histamine might cause the increase in pulmonary capillary permeability reported by Cameron and Sheikh and Tennekoon.

Discussion

From the foregoing, it should be apparent: (1) that there are several factors which may be important in the genesis of pulmonary edema, (2) that opinions vary about the importance of these various factors, and (3) that opinions vary about the mechanisms that produce these various factors.

"Among the dozens of methods employed for inducing pulmonary edema experimentally, there is none which has been proved to act by any other primary mechanism than an elevation of lung capillary pressure relative to tissue pressure."[53] Visscher, in making this statement, is, I believe, probably correct. It should be pointed out, however, that the above statement does not mean that all the methods employed for inducing pulmonary edema have been proved to act primarily by elevation of lung capillary pressure. For example, ammonium ion pulmonary edema has not been shown to be definitely and primarily due to an elevation of the pulmonary capillary pressure. Further, this statement does not mean that an increase in lung capillary pressure will cause edema. Luisada tells that Altschule put a balloon in the left atrium of a dog, sewed up the chest wall, and inflated the balloon. This failed to cause pulmonary edema even though the inflation lasted, in some cases, several hours and must have increased pulmonary vascular pressures.[32]

Once pulmonary edema is started, by whatever primary mechanism (which probably is pressure relationships in most cases), other factors become exceedingly important. As Visscher, himself, mentioned: "to say the capillary pressure rise is a uniform finding . . . does not imply that increased filtration pressure is the only determining factor."[53] Once transudation of fluid into alveolar spaces occurs, the diffusion distances for gas exchange increase and the alveolar ventilation is impaired. Both of these changes lead to anoxia of the alveoli, which Drinker considers to be of utmost importance. Again, once transudation occurs, the effective osmotic pressure in the capillary is reduced. This, of course, favors edema development.

As has been mentioned by Visscher[53] and Cassen and Kistler,[11] it may be that capillary pressure and capillary permeability are not separable but are associated with one another. Visscher mentions the "pores" permeable to protein molecules found in other capillaries and states that it may be that vascular distention under increased pressure has an effect on "pore area" permeable to protein and, consequently, distention itself may render a capillary more permeable. Cassen and Kistler said, "The intracellular cement substance of capillary walls is postulated to consist of processes having elastic properties that tie the walls together."[11] Further, there is a critical value above which the capillary pressure must go before the elastic processes are ruptured and fluid leakage occurs. Capillary pressure may make a capillary more permeable.

Or it may be that increased transudation of fluid can occur in the lungs without the occurrence of any of the above discussed factors, except an increase in lung blood volume. Cournand has demonstrated that a considerable increase in pulmonary blood flow can occur in normal humans without a change in pulmonary arterial pressure.[14] Luisada says, "Hochrein and associates have shown that enormous changes in the volume of capillary bed (in the lung) may occur as a result of altered dynamics, with little or no change in pressure. This is probably due to the negligible resistance to dilatation of pulmonary capillaries due to the absence of significant tissue pressure such as is found elsewhere in the body."[32] The result of an increase in blood volume without pressure changes is an increase in filtering area.[6] This will result in a greater quantity of fluid filtered out of the capillaries.[6] Altschule has suggested this as a factor causing increased transudation of lymph fluid in the lung.[2]

Finally, it should be mentioned that, although one variable such as trauma to the central nervous system or poisoning with ammonium salts may be sufficient to cause pulmonary edema,[35] several investigators have shown that once a tendency toward lung edema exists it may be produced more easily by another factor.[15, 32]

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For example, the Sarnoffs reported that intravenous infusion of 10 cc. of saline per kilogram of dog weight has no effect on the pulmonary venous pressure. But the same infusion, in the presence of marked peripheral vasoconstriction, "produces striking elevations of pulmonary venous pressures."[44] In many cases of pulmonary edema it is possible that a number of factors have been important.

Summary

The complexity of the problem of the pathogenesis of pulmonary edema is evident when the variety of clinical and experimental conditions that apparently cause it are considered.

A decrease in the effective pulmonary capillary oncotic pressure is a factor in the genesis of some cases of pulmonary edema. In most cases this is due to a hypoalbuminemia, but aspiration of fluid with osmotic tension into the lungs may be a cause in other instances.

An "increase" in the negative intra-alveolar pressure occurs on forced inspiration. This may be a factor in fluid transudation is some cases.

An obstruction of lymph flow from the lungs could cause lung edema. The importance of the increase in lymph flow from the lungs that occurs in pulmonary edema is not definitely established.

An increase in the permeability of the pulmonary capillary endothelium will, of course, aid greatly in the production of pulmonary edema. A number of factors have been suggested by various investigators as the cause of an increase in permeability. Some people believe that this is the primary factor in pulmonary edema production.

The current trend of investigators, however, is to incriminate an increase in pulmonary capillary pressure as the primary factor in the genesis of lung edema. Various mechanisms by which such an increase in pressure occurs have been suggested.

It may be fallacious, however, to separate increase in membrane permeability from increase in capillary pressure. An increase in pressure, resulting in distention of the capillary, may make the endothelium more permeable.

The amount of transudated fluid may be increased without any of the above suggested factors occurring. An increase in pulmonary blood volume without an increase in pulmonary vascular pressures can occur. This could cause transudate formation.

Even if one factor is eventually established as primary, this does not mean that it is the only determining factor. Pulmonary edema can be caused by any combination of the above factors. Indeed, once there is a tendency toward edema formation, another factor may more easily cause it.

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BOOK REVIEWS

Clinical Examinations in Neurology. By Members of Sections of Neurology and of Physiology, Mayo Clinic and Mayo Foundation. Published by W. B. Saunders Company, Philadelphia. 370 pages, 76 figures. Price $7.50.

There are many unusual and helpful features about this extremely well illustrated book on the examination of the nervous system. It was designed originally to fulfill the needs of instruction of those training in neurology at the Mayo Clinic, and therefore includes instructions and samples of forms for testing and reporting such phenomena as aphasia, sensory losses, reflex findings, etc.

Electroencephalography and electromyography, speech disturbances, and neuro-ophthalmology receive special treatment in a concise and readable form. The section treating the mental status examination is at times over-simplified and often ambiguous. Thus, for instance, the mood and insight appear to be tested by question and answer technique.

The publication of this manual represents a real contribution to those interested in the examination of the nervous system, both in the training for and the practice of medicine.—*J.A.S.*

Pediatric Cardiology. By Alexander S. Nadas, M.D. Published by W. B. Saunders Company, Philadelphia. 587 pages, 343 figures. Price $12.

These 587 pages devoted entirely to the subject of the heart and its problems in infants and children illustrate the tremendous increase in the scope of pediatric cardiology in the past few years. As recently as 15 or 20 years ago the standard pediatric texts were alloting about 40 pages to the whole subject. The tremendous increase in knowledge and interest has been in the field of congenital heart disease, and it is this portion that receives the most attention in Dr. Nadas' book.

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That knowledge will be gained – and, indeed, the riddle of cancer itself, will one day be solved in the research laboratories. To continue to support this vital work, as well as to carry on its dynamic education and service programs, the American Cancer Society is seeking $30,000,000. We are again appealing to the public to "fight cancer with a checkup and a check."

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tools, acquired heart disease, congenital heart disease, and a short section on anesthesia for children with heart disease. The book covers all of these sections completely and in a readable and informative manner. However, it is the diagnostic tools relating to congenital heart disease, such as radiology, electrocardiography, phonocardiography, cardiac catheterization, and angiocardiography that receive most of the space and attention in that section; and the section on congenital heart disease is approximately half the book.

It is obviously his intention to present a guide to the pediatrician and general physician interested in diagnosing congenital lesions in his own patients. In the foreword, the book is referred to as "a do-it-yourself cardiology." It succeeds admirably in that purpose.—*L.S.*

Proceedings of the Third National Cancer Conference Sponsored by American Cancer Society and National Cancer Institute, Detroit, June 4-6, 1956. Published by J. B. Lippincott Company, Philadelphia. 961 pages. Price $9.00.

This book is unique in several respects. First, because it comprises the verbatim comments and opinions of 200 participating essayists whose names might well fill a "Who's Who" in cancer. Secondly, because practically every major division of the cancer problem is discussed in such a manner that the average interested physician can glean practical worthwhile facts by perusal of any or all of these divisions. Finally, it represents a tremendous bargain in that its 961 pages are replete with readable graphs, tables, and beautifully prepared photographs and photomicrographs.

Contrary to what one might expect from such a conference, this book has achieved a satisfactory balance between a presentation of the theoretical aspects of cancer investigation and the more practical aspects of neoplastic diagnosis and therapy. Actually no more than one-third of the reported proceedings pertain to the purely academic and research phases of the cancer problem, which lends further support to its value as a practical cancer reference source.

In addition to rather detailed discussions on 120 different subjects pertaining to human cancer, there is included a symposium on the end results achieved by treatment. This section deals with results of current cancer therapy employed in the principal types of malignancies involving all of the human organ systems. Also, for the statistically minded physician, there are sections pertaining to survival rates for various types and stages of malignancies ranging from the hemopoietic system to the intestinal tract.

The simplicity of language and yet completeness of discussions pertaining to a vast number of topics without undue verbosity should make this book a worthwhile addition to the library of any practicing physician desiring to keep abreast of current cancer trends.—*D.C.R.*

Physiologic Principles of Surgery. By Leo M. Zimmerman, M.D., and Rachmiel Levine, M.D. Published by W. B. Saunders Company, Philadelphia. 988 pages, illustrated. Price $15.

This book brings to the surgeon something that has long been needed. It is an attempt to correlate various physiologic principles with physiologic problems that present themselves to the surgeon. Dr. Zimmerman, a surgeon, and Dr. Devine, a physiologist, have edited a text that brings together in a lucid discussion the basic principles of physiology as they apply to surgery of the various organs of the body.

For the surgeon, it is an excellent review of physiology, brought up to date. It gives him a correct and concise discussion with the surgical application interlaced. Heretofore, one was required to return to the old physiology textbooks for the physiology of the organs of interest. It should be an excellent book for the doctor preparing for board examination in surgery. Although possibly too specific in its application, it seems to me this book has application for the medical student also.

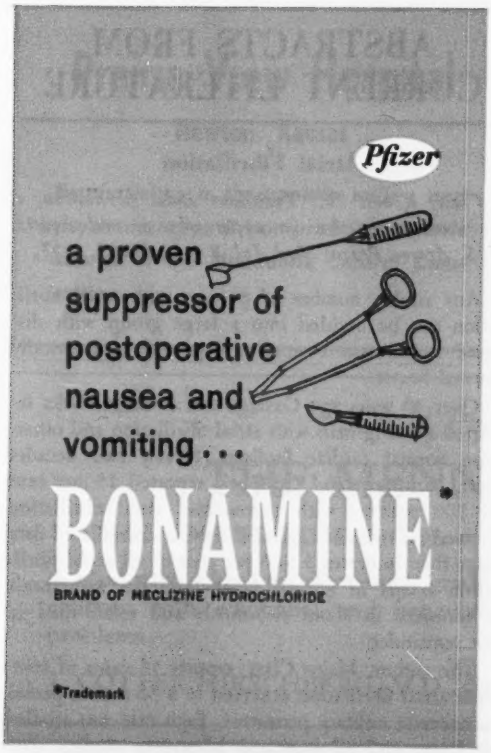
Leading doctors in their fields have written chapters in the book on their particular fields of interest. The editors have attempted to edit all chapters so that wherever possible there will be no overlapping or repetition of knowledge.

This book is highly recommended to the busy surgeon who wants a quick review of the physiology of the organs of the body with their surgical application.—*S.L.V.*

ANNOUNCEMENTS

Annual assembly in otolaryngology, University of Illinois College of Medicine, Sept. 30-Oct. 6. Write Department of Otolaryngology, 1853 West Polk Street, Chicago 12, Illinois.

Ninth postgraduate assembly in endocrinology and metabolism, sponsored by Endocrine Society, Medical College of Georgia, Augusta, Oct. 21-25. Registration limited to 100. Tuition fee $100.



Historical Material Needed

In preparation for the observance of The Kansas Medical Society's centennial anniversary, members of the Committee on History are attempting to collect all material of historical interest. Physicians who can contribute information, records, etc., are urged to send such to

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Approved residencies in physical medicine and rehabilitation available at New York University-Bellevue Medical Center, beginning July 1, starting at $3,400 with dependency allotment added. Apply to Joseph G. Benton, M.D., Institute of Physical Medicine and Rehabilitation, 400 East 34th Street, New York 16, New York.

Sixth annual symposium for general practitioners on tuberculosis and other chronic pulmonary diseases, Saranac Lake, New York, July 8-12, approved for 26 hours of formal credit for AAGP members. Registration fee $40. Information available from Dr. Henry W. Leetch, Symposium for General Practitioners, P. O. Box 11, Saranac Lake.

Eight-week course in occupational medicine, Sept. 16-Nov. 8, offered by New York University Post-Graduate Medical School. Write the Dean, 550 First Avenue, New York 16, New York.

Sept. 1 is deadline for applications for certification by American Board of Obstetrics and Gynecology. Information available from Dr. Robert L. Faulkner, 2105 Adelbert Road, Cleveland 6, Ohio.

Postgraduate symposium on basic sciences related to anesthesiology, offered by University of Pittsburgh School of Medicine, June 10-14, Pittsburgh, Pennsylvania. Registration fee $25. Registration limited to 50. Write 3941 O'Hara Street, Pittsburgh.

Free cardiac surgery program offered by National Jewish Hospital, 3800 East Colfax Avenue, Denver 6, Colorado, to those unable to pay for private care. Applications considered on behalf of patients suffering from cardiovascular defects amenable to surgical intervention. Periodic reports made routinely to referring physician.

Four courses in ophthalmology for specialists offered by New York University Post-Graduate Medical School during first half of 1957-1958 academic year: histopathology, Sept. 16-20; surgery of eye, Oct. 28-Nov. 2; ophthalmoscopy, Nov. 4-8; surgery of cornea, Dec. 2-6. Write Office of Associate Dean, 550 First Avenue, New York 16, New York.

ABSTRACTS FROM CURRENT LITERATURE

Atrial Fibrillation

Class, Robert N.: Transient atrial fibrillation, a frequent occurrence in apparently normal hearts, U. S. Armed Forces Med. Jnl. 8:1-13 (Jan.) 1957.

Any sizable number of persons with atrial fibrillation can be divided into a large group with diseased hearts and a small group with anatomically normal hearts.

Over 40 years ago Gassage and Broxton Hicks reported a young man with atrial fibrillation and otherwise normal cardiac findings. Nearly two decades later, Parkinson and Campbell reported 15 per cent of 200 patients with paroxysmal atrial fibrillation showed no signs of cardiac disease. Accumulated data since then indicates 5 to 6 per cent of all atrial fibrillation occurs in normal hearts and the arrhythmia is transient in about two-thirds and established in the remainder.

The author, Major Class, reports 16 cases of transient atrial fibrillation observed in a 30-month period in overseas military personnel. Each case was studied by x-ray, and all indicated laboratory studies after electrocardiogram tracings confirmed the diagnosis. Pertinent data of each case are presented, and details of four of the cases are discussed. Only one patient was over age 35 when attacks began and in five cases the onset was at an age of 21 or younger. Symptoms included sudden recurrent oppressive and aching precordial pain associated with palpitation and followed by dyspnea. In a 21-year-old airman, attacks had begun at age six, were precipitated by physical exertion or emotional tension, occurred at intervals of a few days to six months and usually lasted only a few minutes, though one had lasted 30 hours.

Hysterectomy in Young Women

Jacobs, Warren M., and Daily, Harold I.: Hysterectomy in young women, Surg., Gynec. and Obst. 102:5 (May) 1956.

This article reviews 68 cases of hysterectomies done on women 30 years of age and younger from a clinical and pathological standpoint.

Tables are included giving the type of procedure performed, indications for surgery, and pathology.

In recurrent resistant pelvic inflammatory disease, the authors feel treatment should be either medical,

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or, if surgery is done, total hysterectomy and bilateral adnexectomy. In a rare instance, the disease may be involving only one adnexa; then unilateral adnexectomy is indicated.

Symptomatic myomas can be treated by myomectomy if technically feasible and if both tubes are patent and other causes of infertility in either husband or wife do not exist.

Patients not desiring more children, who had a prolapse with cystocele or rectocele, were preferably treated by vaginal hysterectomy and repair. Vaginal hysterectomy was used for carcinoma in situ of cervix also.

The authors believe that when a pelvic laparotomy is being done and it is determined that future pregnancies are impossible because of previous removal or bilateral occlusion of the tubes, the uterus should be removed, provided the patient is in good condition and has been informed of such possibility beforehand.

The procedures discussed are offered as a possible improvement over the often-incomplete surgery which has been done in the past and has yielded discouraging results. Further long-term follow-up of these procedures will be necessary for more complete evaluation.—*R.P.S.*

Breast Cancer

Ackerman, Lauren V.: An evaluation of the treatment of cancer of the breast at the University of Edinburgh (Scotland) under the direction of Dr. Robert McWhirter, Cancer 8:5, 883-887 (Sept.-Oct.) 1955.

This article reports the findings and conclusions reached by Dr. Ackerman after a personal examination of the records and pathological tissues of 719 patients with cancer of the breast, which had been previously reported by Dr. Robert McWhirter of the University of Edinburgh. Dr. McWhirter's method of treatment, in all stages of the disease, was simple mastectomy followed by irradiation therapy, which he prefers to radical mastectomy, with or without x-ray.

Dr. Ackerman notes variables which affect the evaluation of such clinical material. These pertain to the judgment and technics of pathologist, surgeon, and radiologist.

Dr. McWhirter feels that x-ray treatment sterilizes cancer at the operative site and in axillary nodes. Dr. Ackerman finds, in numerous pathological sections, that the cancer appears unaffected by previous x-ray therapy.

Furthermore, hormone therapy and x-ray-castration had been used in a large number of instances with an incalculable effect on the course of the disease. There were almost no autopsies, and hence the report of patients "dying of intercurrent disease" is unreliable.

Finally, a persistence of cancer at the primary site in 16 per cent of stage I and II cases was noted; these are the stages which have a high cure rate by radical mastectomy.

Morbidity from x-rays was high in 47 patients, with 3 amputations of the arm.

Dr. Ackerman concludes that Dr. McWhirter's method of treatment can be established only after a much more rigid control of case material. Radical mastectomy, by the trained surgeon, operating on properly selected cases, offers the greatest hope for permanent cure.—*T.P.B.*

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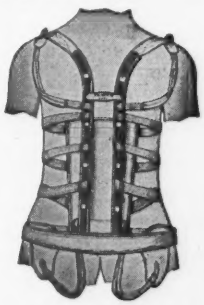

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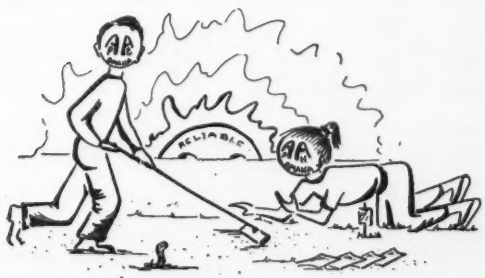

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Ames Company, Inc. ..... Inside back cover
Ayerst Laboratories XII

Bayer Company XVIII
Burroughs Wellcome and Company ..... VIII and 341

California State Personnel Board 349
Central Aircraft Charter Service XVII
Ciba Pharmaceutical Products, Inc. 337
Columbus Pharmacal Company XXIV
Corn Products Refining Company XIX

Fairmount Sanitarium 350
Foot-so-Port Shoe Company 352

Geigy Pharmaceuticals X
Goetze Niemer Company 350

Hanicke, P. W., Manufacturing Company 350

Kansas Oxygen, Inc. XX
Knox, Charles B., Gelatine Company, Inc. XI

Lakeside Laboratories, Inc. V
Lattimore-Fink Laboratories XII
Lederle Laboratories ..... XIV, XV, 310, 311, and 331
Lilly, Eli, and Company XXVI

Medical Protective Company 350
Menninger Foundation 349
Merck, Sharp and Dohme, Inc. ..... XXII and XXIII
Munns Medical Supply Company, Inc. 352

Neurological Hospital 350

Parke, Davis and Company ..... Inside front cover and III
Petro's Surgical Appliances 352
Pfizer Laboratories ..... IX, XII, 339, and 347
Physicians Casualty Association 350
Prairie View Hospital 349

Ralph Clinic XVI
Riker Laboratories, Inc. ..... VI and 343
Robins, A. H., and Company XXI
Roerig, J. B., and Company XIII

Schering Corporation XXV
Searle, G. D., and Company ..... XXIV and 329
Smith, Kline and French Laboratories 354
Southwest Scientific Corporation 350
Squibb, E. R., and Sons 335

Wallace Laboratories ..... Facing XX
Winthrop Laboratories, Inc. ..... XX and 349

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